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## THE INFLUENZA EPIDEMICS OF 1889 AND 1918

You have had the opportunity of seeing much of the present influenza epidemic in our own wards as well as in the various emergency hospitals to which you have been assigned for duty by reason of the general lack of physicians in this community. You therefore know something of the disease as it presents itself at the bedside, and will perhaps be interested to hear from me some account of the epidemic of 1889-90, which occurred when I was a hospital intern, and of the return waves of '90-'91 and '91-'92. A number of additional circumstances have fixed my attention on the disease, so that the intervening thirty years have not eradicated the impressions made upon my mind by the experiences of those early years. It so happened that one of the last medical lectures I heard before my graduation was on influenza, and the lecturer remarked in a somewhat jocular way that he could wish us no better luck than the occurrence of an influenza epidemic soon after our beginning of active practice because it was likely to be wide-spread, all the patients would feel ill and like-to-die, but all would recover, to our great credit. This teaching was based upon many recorded reports of ancient outbreaks; so commonly had the reputation of extreme mildness been given the disease that names like "the delight" were applied to it. The distinguished Benjamin Rush, our first Professor of Medicine, speaking of the epidemic of 1789-90, says that thousands of people suffered in Philadelphia without being confined

to their homes, and remarks that "a perpetual coughing was heard in every street of the city. Buying and selling were rendered tedious by the coughing of the farmer and the citizen who met in the market-place. It even rendered divine service scarcely intelligible in the churches. With few exceptions the malady proved fatal only to old people and to persons weakened by pulmonary complaints, but it carried off several hard drinkers."

This is not the picture of a serious or fatal malady, however wide-spread it might be, and our lecturer, deriving his opinions mainly from the literature and too young himself to have had knowledge of the epidemic of 1848, was quite correct in his teaching. How far, however, the facts differed from the belief was soon brought home to us by the epidemic of the following winter. During the time of its occurrence I was fortunately on duty in the medical wards, and therefore had abundant opportunity for seeing the clinical features of the disease. In addition, I was much engaged in the pathologic department, where we saw many autopsies on fatal cases. I mention these circumstances because of the fact that the hospital records were quite inadequate on account of the number of interns and nurses afflicted by the disease, and that in consequence much of what I have to tell you is based upon my own records and recollection.

In order, however, to give my account additional credibility you must pardon the further personal statement that in 1891 I became associated with Dr. William Pepper, and assisted him in collecting the statistics on which his description of influenza in his "American Text-book of the Theory and Practice of Medicine" was based. This fact possibly more than any other fixed indelibly in my mind the features of the great epidemic of thirty years ago.

I may epitomize my impressions of the main features of the disease in that outbreak as compared with the present one by quoting the words of Hirsch, the great epidemiologist, who said on January 5, 1890: "I am persuaded that the present epidemic differs in no essential particular from those hitherto described.

The great interest which the outbreak of this disease has excited is to be explained simply by the indifference of the great mass of the medical public to everything taught by history. . . .

"Whatever I had to say about influenza I have said in my historico-geographical pathology, and nothing new has presented itself." This somewhat acidulous and opinionated statement is not, after all, far from the truth, as a careful study of the literature will demonstrate.

#### THE PANDEMIC OF 1889-1890

Some time about the first of December, 1889, we began to hear of a great epidemic that had appeared in western Russia, and especially in St. Petersburg, in November. Subsequently accounts of the earlier occurrence—perhaps as early as June—of a similar disease in Turkestan was obtained, but the western world knew nothing of this till the outbreak in European Russia. The first definite report in Philadelphia (*Medical News*, December 21, 1889, and *Public Ledger*, December 21, 1889) referred to the epidemic in St. Petersburg, Moscow, and other Russian cities, and to the appearance of the disease in Paris. The disease was then epidemic also in Berlin, Frankfort, and other German cities, but had not apparently occurred in England. Subsequently those who investigated the matter found probable cases at earlier dates in all these places, and even in this country, where its certain occurrence seemed to have been near the end of December, it was claimed that cases were met with as early as the end of November or the beginning of December. The exact dates of onset have considerable epidemiologic value, but, unfortunately, the difficulty of distinguishing cases of ordinary catarrhal infections from true influenza renders a trustworthy decision as to the character of early cases doubtful. My impression is that the reports of cases in this country prior to the latter part of December are questionable.

The onset of the epidemic in the Philadelphia Hospital was characteristically sudden, and within a few days large numbers of the nurses, attendants, and physicians were attacked. As noted elsewhere then and in the present epidemic, patients in

the hospital were affected in smaller proportion than the personnel of the staffs and the various employees. After a few days, when the disease had become wide-spread throughout the city, cases were admitted from the outside, and at the acme of the epidemic the wards were overfilled and cots were requisitioned to increase our capacity. Many of the later cases admitted to the hospital were pneumonias, and among these the mortality was very high. The appearance of the wards was much the same as that you have recently seen here, but as the epidemic was undoubtedly of milder character and less frequently complicated with pneumonia the excitement and distress in the whole community were far less than in this outbreak. Moreover, the shortage of doctors and nurses in the city, and the undernourishment of the people owing to the necessary food restrictions on account of recent war conditions doubtless increased the public alarm and added to the gravity of the disease. In other essential features it will be found that the two epidemics were identical.

Within a few days over half of our staff of 22 interns were down with fever and the usual symptoms, and an equal proportion of the 100 odd nurses were affected. Apparently the first cases occurred in the nursing staff. Dr. E. P. Davis and Dr. W. S. Vanneman reported (*Medical News*, January 11, 1890) the clinical features of the disease as they observed it among 40 of our nurses. An interesting fact in their account is that the first case in the hospital occurred in an immigrant who had landed three days before at Castle Garden, New York, from whom the disease was communicated to a convalescent patient in the next bed, who during the day acted as nurses' waiter. The disease spread rapidly among the nurses, and in a short time appeared throughout the entire hospital and its population of 3500 persons. The rapidity of its extension was much the same as had been observed in other places. Thus from Paris came the report that at the *Magazin du Louvre* among the 2500 to 3000 employees 300 became ill the first day, and two-thirds of the entire number within three or four days. At the *Pacific Mills*, Lawrence, Mass., among 4242 employees 1699 (40 per cent.) came down with the disease within a few days.

The extent of the epidemic is better shown by subsequent collective investigations made by various governments and individuals than by the ordinary statistics of health boards. It was everywhere reported that the epidemic was a "mild type of malady," and that there was "much discomfort but little fatality," but at the same time the rate of mortality increased enormously in all parts of the world where the disease had made its appearance. Deaths from pneumonia and respiratory diseases generally as well as from all sorts of chronic disease were doubtless due directly to influenza. For example, in Paris, while but 22 deaths were attributed to influenza in one week, there were reported 346 deaths from pneumonia, as against 67 in the same week of the previous year. There were also recorded 131 deaths from acute bronchitis, as against 47, and 138 from bronchopneumonia, as against 27 in 1888. The total number of deaths from respiratory disease was 742, as against 200 in the previous year. This experience in Paris was exactly the same as that in other places in Europe and America. Pepper's statistics included 35,413 cases occurring in the practice of 272 physicians in Philadelphia, but though the general mortality in the city rose to 3044 in January, 1890, when the epidemic was at its height, as against 1488 in December, 1889, only 116 cases were reported as influenza. It is evident that here as everywhere the mortality due to influenza does not appear in the public statistics. Subsequent investigations showed that among all classes of the populations from 40 to 60 per cent. were affected and that the increased death-rate—everywhere doubled or trebled—was due directly to the prevailing epidemic.

The general type of the epidemic was undoubtedly milder than was that of the present outbreak, and the proportion of cases of pneumonia less, but, as I shall show you, the type of the disease and its complications were not at all dissimilar.

The ages of those affected ranged from that of childhood to old age, but it was a notable fact that there was an unusual amount of illness among aged people which was not usually clearly distinguished as influenza, and the death-rate among old people was greatly increased. The major part of the clearly

marked cases of influenza occurred in young adults, but it is altogether probable that the atypical illnesses of the aged were due to the epidemic disease, and for a long time after the outbreak subsided there was an unusual amount of depression of spirits or ill-health among old people.

The nature of the disease had been the subject of considerable discussion and difference of opinion before it reached us on this side of the Atlantic Ocean. The unfamiliarity of clinicians with the disease—owing to the fact that the last preceding epidemic had occurred forty years before—was the principal cause of this uncertainty. Surely none of us who has witnessed either of these great pandemics (1889 or 1918) could be in much doubt if another should occur. In Paris much stress was placed upon the fact that the early cases at least showed practically no evidence of catarrhal infection of the respiratory tract, and on this account some of the leading clinicians believed the disease to be Dengue. Subsequently the respiratory complications and the greater frequency of bronchitis, even in the early stages of the disease, convinced the medical public that the epidemic was really influenza.

Considerable discussion then ensued as to whether the epidemic was an infectious and contagious malady communicated from person to person, or a miasmatic condition attributable to meteorologic and telluric influences. It must be remembered that at this time bacteriology was in its infancy, and nothing was known of the etiology of this disease in particular. It is unnecessary now to recall all of the facts gathered at the time, but it is interesting to observe that in many places the spread of the disease was clearly traceable to contact with affected individuals, and that its incidence was minimal in remote places, such as light-house stations, where contact with people from affected places did not occur. The rapid pandemic distribution of the disease was generally attributed to atmospheric influences rather than to a high grade of infectiousness, though all careful analyses of the chronology of its appearance indicated no more rapid dissemination than could be accounted for by the rate of travel of human beings from place to place.

**Onset.**—The incidence of the disease was characteristically sudden. Perhaps I can best illustrate this by my personal experience. Early in the epidemic I began my usual ward rounds one day at 9 A. M., feeling entirely well. After examining one or two patients I felt slight pains in my fingers as I began to percuss the next patient, and in a few minutes grew chilly and then feverish. Within a half-hour I was too ill to continue my work and was forced to go to bed. My temperature rapidly rose to 104° F., where it remained for two days, accompanied by generalized myalgic pains. At the end of this time the fever and other symptoms rapidly declined, and except for some continuance of bronchitis my attack was completed. Almost at the same time one of my colleagues and my room-mate (Dr. Milner) was attacked, and rapidly developed purulent pericarditis, to which he succumbed in spite of surgical drainage. Others of the staff of interns were seized in equally abrupt fashion, and at one time fully 14 of our number were abed. None, however, was seriously ill, and only the one mentioned died. Of the nursing staff of 100 odd, probably over one-half were affected, but none died. The active epidemic in the hospital did not set in till most of us were convalescent, and the seriously complicated (pneumonic) cases came in when the epidemic in the community was at its height and toward its decline. The same you have no doubt observed in the present epidemic.

Let me now very briefly describe for you the clinical features of the disease as we saw it in 1889.

The symptoms at the onset were general muscular pains, headache, backache, and prostration, the last not so specially severe in the beginning as it became somewhat later. There was in the beginning of the epidemic a rather striking absence of catarrhal symptoms in the earlier stages of the disease which led the Paris clinicians to regard the disease as dengue. Later cases, however, showed increasing prevalence of a catarrhal type with bronchial and pulmonary complications and sequæ well marked.

**Fever.**—The temperature, as a rule, ran quite high in the more acute cases, being from 102° to 105° F., and not infrequently,

especially in the short cases, the decline of fever was a sharp crisis. More often there was a gradual decline of temperature interrupted by recurrences of fever. In many cases after convalescence seemed to be fairly established remissions occurred, and sometimes these continued for a considerable period after the disappearance of all acute symptoms. This was especially so in patients who got out of bed prematurely. There really was no characteristic curve, though the steady rise of temperature followed by three or four days of continued fever, and then a rapid decline to normal, might be regarded as a typical course in acute uncomplicated cases. Cases complicated by pneumonia showed increased fever after the preliminary influenzal period; though many of the most severe pneumonias occurred without marked increase of the temperature.

**Fulminant Cases.**—A number of highly severe cases occurred, especially toward the end of the epidemic. In some of these the patients were suddenly seized with chills, excessive fever, and marked nervous symptoms, and died within twenty-four or forty-eight hours with symptoms and signs suggestive of extreme pulmonary engorgement and without clear indications of pneumonia; others with equal suddenness developed acute pulmonary edema like some cases seen in the present epidemic.

**Pulmonary Symptoms.**—In the beginning, as I have stated, bronchial and pulmonary symptoms were not conspicuous. Later these became more marked, and throughout the epidemic respiratory complications were common. The greatest interest attaches to the question of pneumonia. It is manifest on looking over the records here and elsewhere that many of the cases reported as deaths from pneumonia and other respiratory diseases were actually influenza. The figures I have already given sufficiently illustrate this. In my own service at the Philadelphia Hospital there were admitted very many cases identical with those seen in our wards here during the last few weeks. An interesting feature of these cases as compared with the ordinary pneumonia was the greater degree of prostration, the finely diffuse and usually bilateral bronchitis and pulmonary congestion, and the indefiniteness of the pneumonic physical signs in the areas

presumably pneumonic. For example, in a case with impaired resonance and perhaps a fairly well-marked dulness at one base, the other parts of that lung and the opposite lung would show signs of more or less pronounced bronchitis with dubious indications of patches of lobular consolidation. Frequently, these signs were shifting, so that from one examination to another the whole picture seemed to change. In many of the cases the degree of bronchitis, as indicated by cough, amount of expectoration, and the auscultatory signs, fairly overbalanced the evidence of pneumonia. In such cases the continuance of mucoid sputum and more abundant mucopurulent or sometimes blood-streaked expectoration occurred. In a certain number quite bright blood was brought up. In the unfavorable cases the tendency was in the direction of increasing consolidation, and more particularly when acute toward the development of evidences of pulmonary edema.

A large number of cases of this type died at the Philadelphia Hospital, and I had the opportunity of examining many of these at autopsy. We were struck by the fact that the lesions were almost invariably bilateral and often universal, though one or both of the bases might be specially involved. The solidified portion of the lung on cross-section never showed the dry, granular surface of ordinary pneumonia, but the affected areas of the lung were dark red, even purplish red, the surface on section was moist, and on pressure exuded from the bronchioles a bloody exudate, or in later stages a more or less purulent fluid. The consolidation was rarely complete, and at the same time was not of the mottled type of lobular pneumonia as we were accustomed to see this in the pneumonias of childhood. Among the interns the name "heavy edema" was used because the lung surface exuded a somewhat edematous sanguinolent exudate, and at the same time portions of the tissue sank, when placed in water, in a measure, though not as distinctly, as pieces of the lung in ordinary pneumonia. I had the opportunity of examining the tissue histologically in a considerable number of cases, and found the prevailing features to be enormous engorgement of the blood-vessels, cellular (epithelial and leukocytic) and ery-

throcytic exudation in the alveoli, and infiltration of the alveolar walls. There was little or no fibrinous material in the alveoli, and neither the appearance of ordinary croupous or bronchopneumonia. The outstanding feature was the excessive congestion. There was little pleurisy, but I recall that most of our autopsy material was from cases of very acute character.

**Nervous Symptoms.**—The preliminary depression and neuralgic pains, delirium and other cerebral disorders, and psychoses and peripheral neuritis as sequels were, I think, more common in the epidemic of 1889 as we saw it than in the one that has just passed over. During the acute stage I remember that at the Philadelphia Hospital we had a considerable number of patients more or less maniacal or psychically depressed, so that they had to be transferred to the detention ward. And after the epidemic subsided it seems to me that we saw more instances of paralysis from peripheral neuritis and general nervous depression than have thus far manifested themselves as the result of the present outbreak.

I remember one interesting instance apparently of peripheral neuritis with paralysis beginning in the legs, afterward involving the arms, in which subsequently cerebral symptoms developed and the patient eventually succumbed, with a general picture not unlike that described as Landry's paralysis. Curiously enough, a not dissimilar case with the reverse order has occurred among my patients in the present outbreak. In this instance cerebral disturbances were followed by paralysis of arms and legs evidently due to peripheral neuritis. The cerebral symptoms have cleared up, and the patient is now very gradually improving from the peripheral symptoms.

**Circulatory System.**—I do not recall that anything especially impressed us during the epidemic in so far as the heart and circulatory apparatus were concerned, but, on referring to the literature, I find in the article by Davis and Vanneman, already quoted, that they make note of the fact that the temperature was often 104° F., the pulse rarely exceeding 100. You will see that this corresponds with our experience in the present epidemic. While circulatory depression was not pronounced in the course

of the disease, cardiovascular sequels were particularly frequent, and I recall especially the publications of Sansom, then the leading authority in England, on cardiac sequelæ. He described various forms of cardiac arrhythmia, bradycardia, tachycardia, and the like as consequences of influenza. It is too early at present to predict whether the result of the present outbreak will be similarly marked in the direction of circulatory derangements. It is early enough, however, to state that in the epidemic of 1889 far more cardiac depression resulted from the disease than in the present one, the reason for this being doubtless that at that time older people were to a much greater extent and, perhaps I may say, to a preponderating extent affected.

**Physical Signs.**—In the beginning very few evidences of bronchial or pulmonary disease could be detected in a large number of the cases, as I have already mentioned. In later cases at the onset of the disease the evidences of bronchitis were found, and in the severer cases these were followed by signs of pulmonary engorgement manifested by soft crackling râles and some impairment in the percussion note. Frequently the râles were of a quality that could be best described as "sticky." The breath sounds became increasingly altered, but variable from time to time, and shifting in such a way that it was difficult to determine what the exact pathologic conditions might be. Almost invariably, however, in the most serious cases there developed at the bases of the lungs posteriorly denser and denser consolidation, though not often the complete dulness of an ordinary pneumonia, and throughout the disease the râles persisted. Whether the consolidated and presumably pneumonic portion of the lungs was at one place or another, it was customary to find diffuse signs on both sides of the chest, and shifting pictures of either temporary atelectasis or engorgement, with subsequent relief.

In the most acute of all the cases rapid pulmonary edema developed and, as I have noted before, some patients died before there were any distinct evidences of pneumonia.

Signs of pleurisy and of pleural effusions were not so commonly encountered as in ordinary pneumonia or as we have

seen in the latter part of the present epidemic. I think it but fair to say that our training in methodical physical examination was not as well developed at that time, that we were less prompt to resort to diagnostic needling and, of course, the x-ray was then unknown. For these reasons I surmise that we overlooked many cases of moderate effusions that might be recognized at the present time.

**Gastro-intestinal Symptoms.**—At the onset of the disease nausea, vomiting, and other gastric disturbances were encountered in a certain number of cases, and it was clearly seen that a certain minor proportion of the cases could properly be classified as belonging to a gastro-intestinal variety. In these, disturbances of the stomach, abdominal pains, and diarrhea were among the symptoms, and in some of them these evidences of digestive disorder were pronounced.

**Menstrual Disorders.**—I do not recall from my own experience that menstrual disorders were particularly noted in the epidemic, but I am interested to find in the report of Davis and Vanneman, which was based particularly upon their observations among the affected nurses at the Philadelphia Hospital, that precipitated menstruation was almost habitual. Although they also discuss some cases occurring in the maternity wards, they do not mention premature labor or abortion. Reference, however, to the older literature reveals the fact that for a long time it had been recognized that influenza tends to bring about these results in pregnant women. Curiously enough, this fact had been little recognized prior to 1889, and had been again forgotten between that time and the present epidemic.

**Complications.**—Among the complications, in addition to the nervous symptoms I have already mentioned, there were a certain number of cases with suggestions of meningitis or actual meningitis, though we did not at that time practice the method of lumbar puncture, later introduced, which might have enabled us to diagnose the condition more certainly.

The most frequent and outstanding complications were those affecting the lungs and pleura. I have indicated sufficiently the frequency of pneumonia. Pleural effusions and

empyema occurred in a relatively small proportion of cases, but persistent consolidations of the lung and long-continued bronchial troubles were met with as sequels very frequently.

Cardiac and cardiovascular complications and sequels were not at all infrequent. During the height of the epidemic pericarditis now and then developed.

Renal complications were apparently infrequent. I cannot give any figures as to the frequency of albuminuria and cylindruria, but it is quite certain that very few cases of actual nephritis developed.

Among the sequels of the disease, persistent asthenia, lack of vitality and resistance, a general depression of the nervous system, and a tendency to coldness and inability to bear lowering of temperature were very marked. This also I believe may have been more conspicuous in the earlier epidemic than in the present on account of the larger number of people of advanced years affected in the outbreak. I recall particularly cases in which for months and even for years after the attack patients were in a reduced state of vitality that made them susceptible to every change of temperature, and forced many of them, fortunate enough to be able to do so, to seek warm climates with the beginning of cold weather.

**The Bacteriology of Influenza.**—At the time of its outbreak in 1889 nothing whatever was known regarding a possible specific organism, and not until 1892 was any light cast upon this subject. Pfeiffer, however, succeeded in isolating a Gram-negative bacillus of very small size which in its staining peculiarities suggested a diplococcus, and later this organism was accepted by most authorities as probably the specific cause of the disease. After the subsidence of the great pandemic and the return epidemics of 1890 and 1891 the influenza bacillus was commonly found by bacteriologists in endemic cases, and it seems certain that it had never wholly disappeared before the outbreak of the recent recurrence. I shall refer to it again after speaking of the present epidemic.

THE SECONDARY EPIDEMICS OF 1890-91 AND OF 1891-92, AND  
THE PSEUDO-INFLUENZA OF SUBSEQUENT YEARS

Like a number of the great pandemics recorded by medical historians, the epidemic of 1889-90 was followed by return waves during the two following winters. In this respect it resembled especially the epidemics of 1729-30, 1788, 1830, and 1848, all of which were followed by secondary outbreaks. The return epidemics of 1890-91 and 1891 and 1892 were in some places, as in Great Britain, more extensive and severe than the first, and in practically every part of the world where the disease appeared during the original outbreak some degree of recurrence was observed. Even before these definite recurrences of the two following winters there had been in some places minor return waves of the disease during the spring and fall of 1890. After 1892 repeated fall and winter epidemics of catarrhal infections occurred, and these were at first reported as influenza and later were more generally spoken of as grip—no doubt with the subconscious thought that though the name *la grippe* was originally used as synonymous with influenza, the English equivalent did not altogether commit the reporter to the diagnosis of true influenza. Taken individually, cases in these so-called "grip epidemics" could not be distinguished from the milder cases of true influenza observed in the great epidemic, but taken as a whole the recurring winter epidemics did not impress those of us who had seen the great pandemic as being the same disease. After Pfeiffer's bacillus was discovered bacteriologic studies of these minor epidemics repeatedly showed abundance of streptococci, pneumococci, or *Micrococcus catarrhalis*, but only exceptionally the influenza bacillus, and names like "pseudo-influenza" or "influenza nostras" were suggested for these influenza-like outbreaks. It is perhaps unnecessary for me to detail the clinical differences, but, in brief, I think it worth while to direct your attention to some of the main facts. The pseudo-influenza cases, as a rule, show a greater tendency to produce catarrhal affections of the nose and throat (coryza, sinusitis, tonsillitis, membranous deposits in the throat, and uncomplicated bronchitis); the depression of the patient is far less con-

spicuous, and cyanosis and other toxic manifestations are less evident; leukocytosis is common, gastro-intestinal symptoms and neuritis or other nervous conditions less frequent, while pneumonia is far less common, and when it occurs more distinctly an ordinary pneumococcic complication, with the usual physical signs and clinical features. The mortality of the epidemics as a whole is comparatively negligible. If we include in the group under consideration certain epidemics of streptococcus infection (milk infections) of recent years, and the outbreak of last winter in our cantonments, the last statement regarding mortality would, of course, require modification, but the clinical features would nevertheless distinguish these epidemics from true influenza. For several years past in filing our histories of patients in the University Hospital we have adopted the title pseudo-influenza to designate these cases. Perhaps we may in the future be able to separate them into defined groups, but for the present no better nomenclature seems to offer itself.

Incidentally, I think it of some importance to direct your attention to the fact that the recurring winter epidemics have given physicians as well as laymen the impression that influenza is always a winter disease. Almost the contrary appears to be the case. At all events, a number of the great pandemics of the past, including the present one, began in the early fall or even in late summer, and on account of this misapprehension doubt was expressed regarding the nature of the recent outbreak when it was first reported in Europe.

While the diagnosis of individual cases is uncertain, that of epidemics as a whole ordinarily should not occasion great difficulty. The greatest cause of confusion lies in the fact that after every pandemic outbreak residual cases no doubt act as carriers and occasion endemic recurrences, and even mild epidemics follow.

#### THE PANDEMIC OF 1918

During the summer of 1918 reports came to us that there had appeared in Spain a wide-spread epidemic of influenza of some-

what virulent character. Later the disease appeared among the Austrian and German armies; still later reports came to us from our own armies and those of our Allies in France. In extent and in the seriousness of the cases the epidemic seemed to equal or surpass that of 1889, and from the rapid pandemic distribution, as well as from the character of the symptoms reported, there was little doubt but that this so-called Spanish influenza was the same disease as the "Russian influenza" of 1889. Not until early or mid-September did the disease reach this country, and its outbreak here was attributed to the arrival at Boston harbor of a ship bearing numbers of sailors ill with the disease.

It may be interesting, however, for me to refer to the fact that we had during the previous month in our wards here a curious epidemic condition among Hindu sailors landed from a British ship arrived in this port from Liverpool. Some 30 Hindus and 1 white man, a petty officer, on board the ship were the patients received by us. All of them had been taken sick five days or more after the ship had left Liverpool, where there was at the time an epidemic of bronchial or pulmonary infection mainly among the seamen. I shall not pause now to point out the differences in the cases of these Hindu sailors from those met with in the influenza epidemic that broke out a month later, but shall merely mention the facts that they all were suffering with unusually violent bronchial catarrh with abundant mucopurulent expectoration, that they developed a form of pneumonia with curiously disseminated patches of consolidation, that the blood-picture was that of a marked infectious leukocytosis, and that none of them showed any evidence of pleural complications. The respiratory rapidity and the marked dyspnea, the rapidity of pulse, the leukocytosis, and in the fatal cases the occurrence of small foci of suppuration in the lung distinguished these cases very sharply from true influenza. Our bacteriologic studies showed cultures of *Micrococcus catarrhalis*, streptococci, and pneumococci, but no influenza bacilli. I do not, of course, pretend to say that the last organism was not present, for the difficulty of its culture to those who have not previously had experience with the epidemic disease was well recognized; but

the symptoms and pathology of the disease marked it as something different from influenza.

**Onset of the Influenza Epidemic.**—The epidemic apparently made its appearance in this country at Boston Harbor, whence it rapidly spread to the people of the city, and later to adjoining portions of the State of Massachusetts. Subsequently it appeared in rapid succession in most of the important centers of population in the East. Its appearance in the middle and far West and finally on the Pacific Coast was so timed as to suggest the transportation of the infectious agents by affected individuals.

The first reports of the disease in Philadelphia came from the Naval Station at League Island and the Naval Hospital, and the epidemic became extremely severe in these centers before the community as a whole began to be affected. After an interval of ten days or two weeks it appeared in all parts of the city and rapidly assumed gigantic proportions.

I shall not attempt to detail the symptomatology of the disease, as you have seen it, but wish merely to contrast its features with those of the earlier epidemic which I have described for you, and to allude to some of the striking or peculiar features of the present outbreak.

The onset of the disease was much like that in 1889, although the nervous symptoms seemed to be less marked and the catarrhal symptoms (bronchitis) more pronounced. One striking symptom that occurred in many of the early cases was cyanosis. Sometimes this was of mild degree; at other times profound, despite the fact that circulatory weakness and even marked respiratory embarrassment or extensive bronchial involvement were not present. The cause of this cyanosis has not been clearly determined. There is a disposition to attribute it to changes in the blood itself, to methemoglobinemia, but I cannot say that this is established. Certainly it was not attributable to circulatory causes. In the latter part of the disease when pneumonia entered in as a complication another type of cyanosis frequently presented itself, though perhaps no more commonly than would be the case with ordinary pneumonias.

Cough and catarrhal symptoms were certainly conspicuous from the beginning in the great majority of cases, and in this particular again the disease differed from that of 1889. Abundance of sputum, sometimes merely mucus, more frequently mucopurulent, was usual; but in many cases there was a dark brown (prune juice) expectoration or it was bright red from admixture of blood. Occasionally there was free hemoptysis.

**Respirations.**—The patients frequently presented little embarrassment of respiration despite excessive fever, and even when pneumonia developed; and sometimes with excessive bilateral pneumonia the absence of respiratory embarrassment was conspicuous. Perhaps the majority of the cases showed no decided increase in the rate of breathing until the pneumonia had reached its latest stages.

**Physical Signs.**—These were practically identical with those seen in the earlier epidemic, except that they developed more promptly, and were, on the whole, more conspicuous. There was, however, the tendency to diffuse bilateral involvement, with evidence of a distinct localization at one or both bases, or sometimes the apices. The shifting character of the physical signs was much the same, and the long-continued residua of bronchitis, consolidation, and perhaps pleural exudation exactly reproduced conditions as I had seen them before. There were cases in which the probability of bronchopneumonia could scarcely be doubted, though the physical signs gave little evidence. There were others in which the probability of pleural effusion seemed undeniable, though needling and x-ray examination failed to detect any exudate, and there were cases in which the shifting character of the signs from day to day left one in complete doubt as to the pathologic lesions present.

**Cardiovascular Apparatus.**—I have mentioned to you the fact that the relative infrequency of the pulse was noted in the former epidemic, but it certainly was far less striking than in the present, since few writers allude to it. I do not recall that we commented on it particularly at the time. Among our recent cases it has been so marked a feature that one could not well escape observing it, and from the tables I shall show you you

will see how very pronounced this feature was. In not a few of the cases even when extensive pneumonia had developed the pulse-rate remained below 100, 90, or even 80.

During the height of the epidemic but little cardiac arrhythmia or other forms of cardiac disturbance were observed. Toward the end of the epidemic, however, some cases of this type made their appearance, and many other conditions—bradycardia, tachycardia, extrasystolic irregularity, and auricular fibrillation—occurred. Mild cardiac pains were not uncommon. Severe anginal attacks were rare. A number of cases of syncope, however, occurred, and in one at least I observed a fatal syncope in a convalescent patient who showed no objective evidence of cardiac disease on careful examination after the first and rather mild syncopal attack.

**Gastro-intestinal symptoms** were, perhaps, more common during the present epidemic, though cases of the so-called gastro-intestinal type were possibly more rare. A number of cases of vomiting of blood, a few instances of hemorrhagic diarrhea, occasionally excessive and exsanguinating, were met with. Toward the end of the epidemic it seemed to me that gastro-intestinal symptoms became less common.

**Renal Manifestations.**—Albumin and casts were very commonly found in the urine, and seemed to me of little significance except when they were accompanied by other evidence indicative of a nephritis. A few cases of acute hemorrhagic nephritis with pronounced uremic symptoms were observed in our series.

**Nervous Symptoms.**—Headache, backache, pains in the extremities, and various forms of neuralgia marked the onset of many cases. Though severe cerebral symptoms were rarely seen in the early stages of uncomplicated cases, I understand that the experience in some of our army camps in France was very different. There, I am told, intense headache and signs of meningeal irritation were not uncommon, and frequently required lumbar puncture for their relief.

In the later stages of the disease, depression, apathy, and loss of muscular power were occasionally found; paralyzes occurred in some cases, while peripheral neuritis and local paralysis from

whatever cause occurred only occasionally. The present epidemic is not greatly different from the earlier one so far as the nervous symptoms are concerned, though, on the whole, it has seemed to me these symptoms have been less marked. Among more severe symptoms I have observed stupor and convulsions apparently independent altogether of any renal complication.

**Menstrual Functions.**—Precipitated or premature menstruation was so habitual a symptom that it was regularly expected by us in all women patients, and you have heard the danger of abortion or miscarriage so frequently emphasized that I need not do more than mention it now. The particularly high mortality of the disease in pregnant women was everywhere commented on.

**Special Sense Symptoms.**—Disturbances of vision, of hearing, and of the olfactory sense have occurred among a small number of the cases, but seem to have been definitely less frequent than in some of the epidemics of pseudo-influenza of other years. These symptoms referable to the special senses were about as common in the present epidemic as in 1889.

**Complications.**—I have already mentioned in connection with the special organs complicating conditions that have arisen, and wish now only to refer again to pulmonary conditions. It was very striking that in the early period of the epidemic pleural complications were comparatively rare, while severe pneumonias, often rapidly fatal, and sometimes overwhelming pulmonary infection, with fulminant pulmonary edema, gave the disease its most striking characterization. Later, pleural complications came to be more and more in evidence, partly because some of the pneumonia cases dragged on and the later-occurring influenzal infections apparently were less virulent and allowed time for the development of pleurisy. It was conspicuous, at all events, in our wards toward the end of the epidemic that the mild influenzas had all recovered, that the acute cases of pneumonia had recovered or died, and that we were left with a group of cases of residual unresolved pneumonias, empyemas, and cases of pleural thickening. It is from this group, no doubt, that the large numbers of chronic bronchial and pulmonary sequels of

influenza are recruited. It was a noteworthy fact that after the epidemic of 1889 pulmonary disease seemed to have increased enormously throughout the world, and in the light of our present experience, with our better means of investigating cases by the methods of bacteriology and radiography, it is possible to assert that many cases of supposed tuberculosis of the lungs have undoubtedly been instances of postinfluenzal residua.

Among the lesions present in these cases, in addition to the ones I have already mentioned, bronchiectasis should be remembered. The circumstances are such as to make the development of this condition altogether likely; for, as in the bronchopneumonias of childhood, neighboring bronchial tubes become weakened and dilated, so in the influenzal pneumonias of earlier or later life the same thing takes place. Certain it is that many individuals date chronic bronchial disease (possibly or probably bronchiectasis) from a severe attack of influenzal pneumonia with delayed and incomplete resolution.

**Bacteriology.**—Studies of the bacteriology of the disease were prosecuted with unusual zeal in various places, and though a final decision cannot be expressed regarding the etiology of the disease, some facts of importance were brought out. A Gram-negative bacillus answering closely to the original description of Pfeiffer was obtained from the sputum of a considerable proportion of the cases studied. In some laboratories it was found in as many as 80 per cent. of all cases studied—elsewhere smaller proportions of positive cultures were reported—a discrepancy that may be accounted for by some of the technical difficulties and by unfamiliarity with the best methods of cultivation. In a high percentage of the cases in which cultures were made from the lungs at autopsy the same organism was recovered; and in a small proportion of cases blood culture was reported as positive. Associated with this organism the sputum and lung cultures showed streptococci, the pneumococcus, Friedländer's bacillus, and occasionally various other organisms. The frequency of occurrence of the so-called influenza bacillus contrasts very strongly with bacteriologic experience in any of the recent epidemics of acute respiratory disease, such as those

of the winter of 1917-18, as well as in previous outbreaks of similar kind, and suggests the possibility of an etiologic relationship of the bacillus to the disease. Moreover, the clinical features of the disease were not such as would correspond with infection by any of the associated organisms named. The possibility of the disease being due to a filterable virus was suggested by some of the features in the epidemiology, but direct investigations gave no support to this assumption. At present, therefore, probability seems to lie in favor of the influenza bacillus as the cause of the epidemic, with other organisms of importance in certain of the cases complicated by pneumonia or other conditions. There is, however, abundant ground for believing that mixed infection is not at all essential to the occurrence of pneumonia.

**Cause of Death.**—One of our cases represented the type of acute fulminant influenzal death. The patient, one of the pupil nurses, was on duty and apparently well till 7 P. M., when she reported to her head nurse that she felt feverish. Her temperature was 100° F. During the night her fever increased and she presented physical signs of pulmonary congestion. Early in the morning pulmonary edema developed, and at 11 A. M. the patient died, without having developed clear evidences of pneumonia. Excepting in this case physical signs were found in every one of the 52 fatal cases which had led to a diagnosis of pneumonia before the death of the patient. In some of the cases these signs were indefinite and scattered, but in none were we in doubt as to the actual presence of pneumonia.

Some of the statistics of our hospital cases will interest you:

**I. Cases:**

Influenza, uncomplicated.....	150		
Influenzal pneumonia (56 per cent.).....	113—died, 52 (46 per cent.)		
<hr/>			
Total.....	263—	52 (20 per cent.)	
Cases admitted with pneumonia.....	84—	46 (54 per cent.)	
Cases developing pneumonia in hospital....	29—	6 (20 per cent.)	

You will note that a large percentage of our cases were admitted to the hospital with pneumonia, and many of these were practically moribund and lived only a few hours.

## II.—*The Ages of the Patients:*

15 to 20.....	24
21 to 30.....	105
31 to 40.....	41
41 to 50.....	7
51 +.....	5

The ages were almost exactly the same in the uncomplicated cases and the pneumonic group.

### III.—Symptoms at Onset of Influenza:

Pains in the head, back, limbs, or abdomen occurred in about 60 per cent. of all the cases.

Gastro-intestinal symptoms—nausea, vomiting, or, more rarely, diarrhea—occurred in 25 per cent.

Cough and mucoid expectoration were present in 75 per cent.

The onset of fever was abrupt in 85 per cent.

Cyanosis was frequent but marked in but a small number. The figures recorded are uncertain.

#### IV.—*The Pulse-rate:*

In uncomplicated influenza the average rate was 75 to 85 or 90; few cases (6 or 8) 100 to 105.

In pneumonic cases the average pulse-rate throughout the disease was 100 or less in 80 per cent. Toward the end of fatal cases the pulse-rate rose over 100 in less than 50 per cent.

### V.—The Respiration Rate:

In uncomplicated influenza the highest rate was 31; in practically all the rest it was from 20 to 23.

In the pneumonia group we find the following figures:

Recovered cases, above 40.....	1 case (late)
above 30.....	23 cases (early)
	8 " (late)
above 20.....	28 " (early)
	37 " (late)
below 20.....	5 "
Fatal cases, above 40.....	13 " (early)
	18 " (late)
above 30.....	24 " (early)
	14 " (late)
below 30.....	9 "

The respiration rate was, therefore, a better indication than the pulse of probable fatality, but was by no means a trustworthy guide.

### VI.—*The Temperature:*

In uncomplicated influenzas the highest (average) temperature was from 100° to 102° F. in the great majority of cases. Occasional elevations to 104° F.

occurred, but an average height of 104° F. was not shown in any of this group.

In the pneumonic group there were—

19	cases	showing	average	104° F.
34	"	"	"	103° F.
23	"	"	"	102° F.
9	"	"	"	101° F.
7	"	"	"	100° F. or less.

VII.—*The Blood-count:*

	Uncomplicated cases.	Pneumonic cases.
15,000 + .....	3	12
9-15,000.....	13	21
2- 9,000.....	38	26

VIII.—*The Urine:*

In uncomplicated influenza albumin and casts were found in 28 cases, and either albumin or casts alone in 14.

In the pneumonia group albumin and casts were reported in 49 cases.

IX.—*Onset of Pneumonia:*

The time of onset was within the week after beginning of influenza in 42; over a week in 17; doubtful in 40. Chills marked the onset in 22; sudden increase of fever in 27 (in 16 of these there was no chill), and by change in physical signs in 18.

X.—*Termination of the Pneumonia in Recovered Cases:*

Decline of fever by lysis in 38.

Decline of fever by crisis in 13.

XI.—*Physical Signs of Pneumonia:*

Definite.....	75
Indefinite.....	18
Doubtful.....	6

The terms "definite," "indefinite," and "doubtful" are here used in the sense of the evidence of distinct areas of consolidation. In the doubtful cases there were auscultatory signs, leaving little question of the presence of pneumonia.

XII.—*Distribution of the Pneumonic Areas:*

<i>Definite:</i>		<i>Indefinite and doubtful:</i>	
Lower lobe, Left.....	21	Right lung.....	7
Right.....	16	Left lung.....	4
Both.....	26	Both lungs.....	5
Upper lobe, Left.....	1	Scattered areas.....	8
Right.....	0		—
Whole lung, Left.....	2		24
Right.....	3		
Upper lobe and opposite base..	6		
	—		
	75		

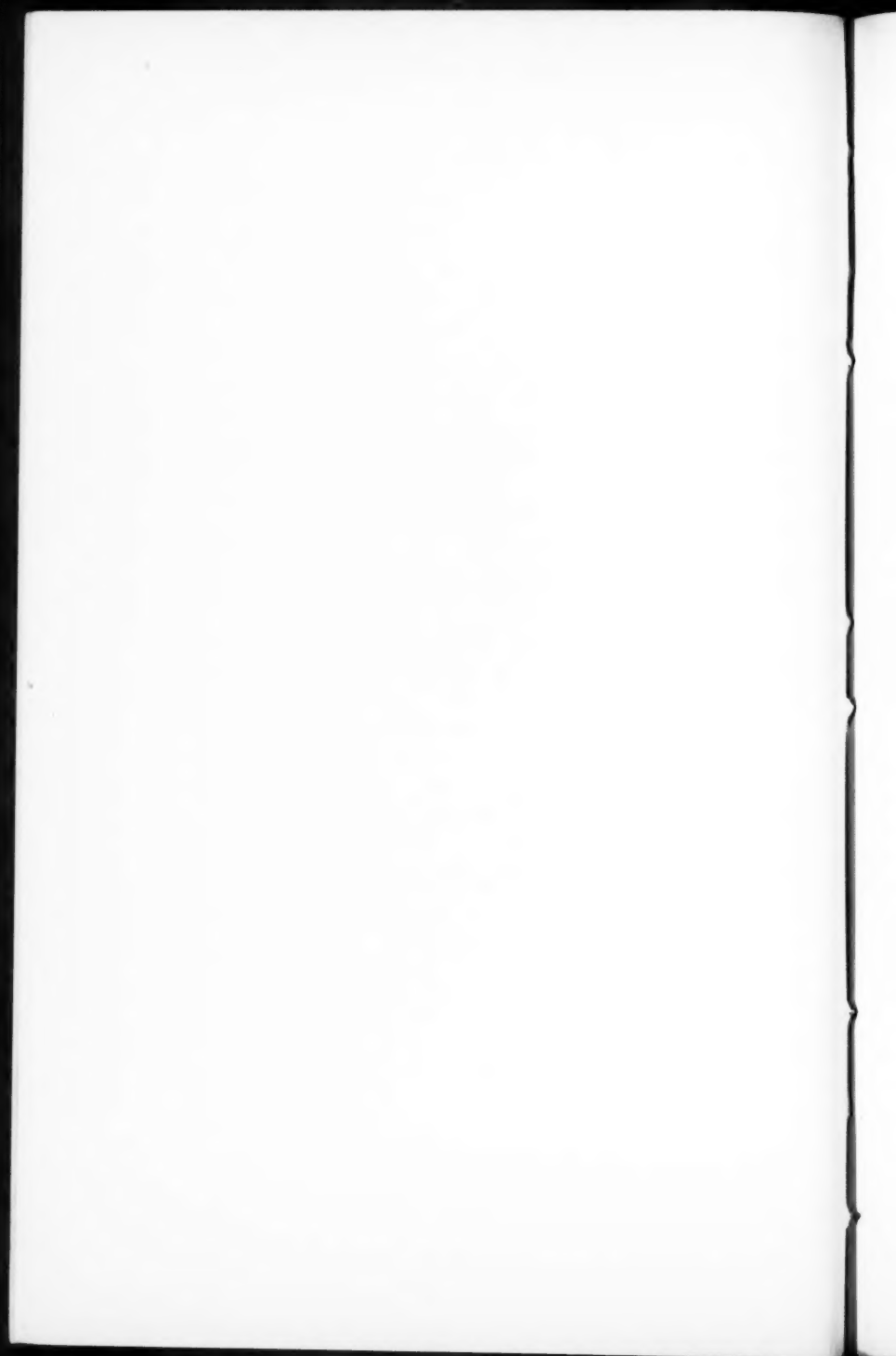
XIII.—*Marked Cyanosis:*

Fatal cases.....	17
Recovered cases.....	3

XIV.—*Pleurisy:*

Indicated by physical signs or x-ray.....	22
Confirmed by needling.....	4

In conclusion, let me call your attention to what you probably observed in the course of the epidemic—the variation in the clinical picture of the disease at different periods of the outbreak. In the beginning, short cases recovering quickly or severe ones terminating in rapidly fatal pneumonia without complications were conspicuous. Later the cases were less acute and pleural complications began to become more frequent. Toward the end, as at present, our wards have contained numbers of influenzal and pneumonic residua—pleural thickening, empyema, unresolved pneumonia, etc. In all these particulars the present epidemic does not differ from those of the past; and it is safe to predict that sequels such as neuroses, peripheral neuritis, sinus infections, general asthenia, and cardiac disorders will follow, and return waves of minor epidemics will make their appearance. The number of cases of pulmonary residua with dubious physical signs will make the diagnosis of pulmonary tuberculosis in the absence of discovered bacilli more questionable than before the epidemic made its appearance.



## CLINIC OF DR. H. R. M. LANDIS

### UNIVERSITY HOSPITAL

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#### INFLUENZA AND SOME OF ITS COMPLICATIONS

THIS morning I wish to discuss with you some of the phases of the present epidemic of influenza as we have seen it in the wards of the University Hospital. So far as I have been able to gather from reading the accounts of past pandemics, the present one differs little from those that have been recorded in the past except that, possibly, the number and the severity of the complications have been greater. While we have had a few instances of the nervous and of the gastro-intestinal type of the disease, the bulk of the cases have belonged to the respiratory type. The severity ranges all the way from the simple, benign influenza of two or three days' duration to those with serious and fatal complications.

The history of the onset as given by the patients is singularly uniform. The attack may develop with malaise and drowsiness, chilly sensations, to be followed by headache, pain in the eye-balls, and pain, often very intense, in the bones, muscles, and small of the back. In addition, in the respiratory form, there is commonly coryza, sore throat, soreness or actual pain in the chest, and cough, which at first is apt to be unproductive and later accompanied with mucopurulent sputum. The sputum, in addition, may be blood-tinged and at times consists of almost pure blood. Gastro-intestinal disturbances are relatively common for a day or two, but only in exceptional instances can they be looked upon as severe enough to consider the case one of the true gastro-intestinal type. Many of the patients complain of nausea and vomiting for the first few days of the attack. While it is said in the previous epidemics that a mild type of the disease is sometimes seen in which there is little or no fever, none of this type

has come under our observation. Fever is nearly always present, and may at the onset be quite high. The temperature chart may show a high continuous type of fever for a few days, or it may

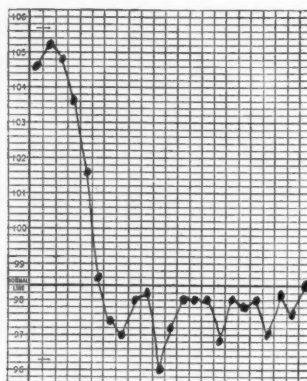


Fig. 127.—Simple influenza. Fever of short duration, with sharp decline.

rapidly fall to normal in the course of thirty-six to forty-eight hours (Fig. 127). More often it gradually falls by lysis (Fig. 128). Often the temperature falls irregularly, and at times the

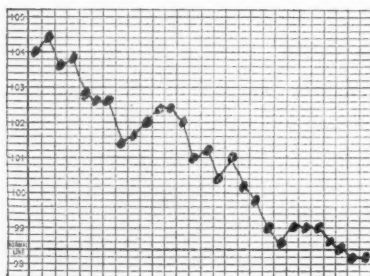


Fig. 128.—Simple influenza. Fever of four days' duration. Gradual decline. A few rales at both bases. No other physical signs.

fever curve may be marked by sharp intermissions (Fig. 129). Not uncommonly when the fever subsides the temperature may

become subnormal and may continue this way for days and often weeks (Fig. 130). During this period a marked slowing of the heart action is common, the pulse-rate dropping to the 50's or 40's. Several of these cases have been studied by means of the

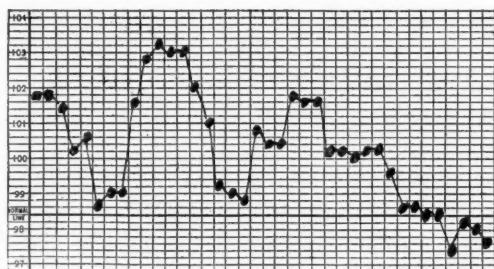


Fig. 129.—Simple influenza. Eight days' duration. Marked congestion of pharynx and tonsils. Gastro-intestinal symptoms for two days. A few scattered râles at bases of lungs.

electrocardiograph, but nothing abnormal was found. The respiratory rate even in uncomplicated cases is subject to marked variations. It may be quite high, but rarely continues so unless there is complicating lobar or bronchopneumonia. In the simple

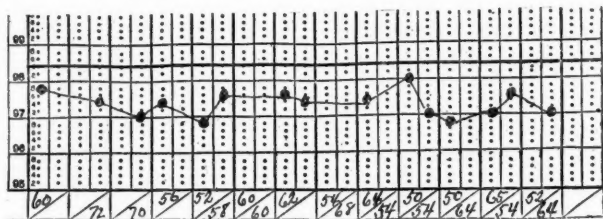


Fig. 130.—Subnormal temperature and low pulse-rate during convalescence from influenza.

uncomplicated case it may range from normal to 30 or 40 times a minute. The explanation is not clear. Graves attributed the high rate to inhibition of the pneumogastric nerve, while Lichtenstern thought it might be due to marked but transient

congestion of the lungs. Possibly it may be an evidence of acidosis. As I have seen these cases I have come to fear a high respiratory rate much more than an unduly rapid pulse or high fever. Patients with a high respiratory rate always seem to be extremely toxic.

Profuse sweating is also noted in many of the cases, and is often marked even in the presence of very high fever.

While a few cases of the milder type will show little or no evidence of bronchitis, the great majority will, on examination, reveal the presence of râles over both lungs. The râles may be distributed fairly evenly on both sides, but in most instances they are most marked and commonly limited to the bases posteriorly. The râles are usually fine and sticky in character, and in many instances the fine, crackling sound close under the ear suggests involvement of the pleura as well. When the involvement of the respiratory tract is limited to involvement of the upper tract and bronchitis, the duration of the attack is from two to six or seven days. The temperature then falls to normal, pain disappears, and the râles have either disappeared or rapidly diminished from day to day. On the other hand, if the chest condition is protracted beyond a week, or even after the fever has disappeared the râles persist, there has probably been some bronchopneumonia. This is almost certainly the case if dyspnea is at all marked and the patient is prostrated. I will speak of bronchopneumonia more in detail later.

In a few instances we have seen what was apparently a relapse of the influenza infection without serious implication of the lung itself. In such cases after a febrile period of three or four days the temperature falls to normal or nearly so. After a few days the temperature again rises for a brief period (Fig. 131). If the second febrile disturbance is prolonged, however, it is more than likely that this secondary rise has been the result of some intercurrent disturbance, either of bronchopneumonia or a small collection of fluid. This has certainly been our experience as a result of x-ray examinations of these cases.

Shortly after the last great pandemic of influenza Pfeiffer, in 1892, discovered the influenza bacillus, and ever since that



etc. The streptococci and pneumococci are by far the most important, and to them is to be attributed most of the serious complications.

In the respiratory type of the disease the entire respiratory tract is involved to a greater or less extent. The tonsils, pharynx, larynx, and trachea as far down as can be seen are inflamed. At the autopsy table the bronchial mucous membrane is more or less congested and inflamed and often filled with a sanguinolent, frothy mucus. In addition, the bronchial wall is swollen and softened. It is quite likely that this predisposes the bronchi to dilatation. It has long been recognized that there is a close connection between influenzal infection and bronchiectasis, and that the bacillus of influenza is the one organism most frequently associated with this condition.

In the present epidemic, so far as we have observed it, but relatively few of the cases were of the simple uncomplicated type. The occurrence of bronchopneumonic areas was extremely common. These areas are in some instances small and located in the lower lobes near the hilus, or they may involve very considerable portions of the lower lobes.

Bronchopneumonic areas occurring in influenza often show distinct interstitial changes, and it is quite likely that this accounts for the persistence of these areas of consolidation and their slowness in resolving.

In some cases the lobar involvement is typically of the pneumococcus type. In others the lobar involvement is atypical. It often presents the appearance of being lobular primarily and only gradually becoming lobar. Thus there will be seen lobular areas in various stages.

Influenza pneumonias, whether bronchial or lobar, seem to be complicated by effusion more often than the ordinary types of these affections. The fluid may be free in the pleural cavity or encapsulated. Usually these effusions are purulent, and their presence should always be suspected when the fever is unduly prolonged.

At times in these epidemics a very fulminant type of the disease is encountered in which death occurs early. At the

autopsy the lungs are found to be greatly engorged and at times show the presence of hemorrhages into the pulmonary tissues.

### BRONCHOPNEUMONIA

The first complication which I wish to discuss with you is bronchopneumonia. The condition is present in such a large number of the cases that it is not altogether clear as to whether it should be considered as a complication or an integral part of the disease. Owing to the paucity of physical signs in many of the cases it escapes detection. Those of you who have been interested, during the height of the epidemic, in studying the causes of death as reported in the daily papers, must have been struck with the frequency with which death has been ascribed to influenza. From what I have seen of the cases which have been very ill and especially those which have died, I am convinced that bronchopneumonia or lobar pneumonia is always present, and that when influenza has been assigned as the cause of death it has been a mistake.<sup>1</sup>

How are you to determine the presence of bronchopneumonia? The 2 cases which I am about to discuss with you are excellent illustrations.

CASE I.—This patient was admitted to the hospital complaining of dyspnea, cough, and soreness all over the body. The illness had started a week previously. At that time he felt chilly, had some fever, sweating, headache, and cough. Later the cough became worse and he expectorated a considerable amount of brownish mucus, which was also streaked with bright red blood. At the beginning of the attack he was nauseated and vomited a good deal.

A glance at his temperature chart (Fig. 132) shows that for the first few days his fever was of an irregular type, but was gradually declining by lysis. It did not become normal for more than a day, however, when it again rose, was irregular for a few days, and finally subsided. We felt in this case that something additional had developed at the time of the second febrile dis-

<sup>1</sup> Since making the above statement Christian (Jour. Amer. Med. Assoc., November 9, 1918) has directed attention to the same point.



turbance. An examination of his chest yielded the following information: Posteriorly on the right side near the angle of the scapula and in the immediate neighborhood of the interlobar septum there was a patch of dulness about the size of the palm of one's hand. Over this area there was distant bronchial breathing and marked transmission of both the spoken and whispered voice.

The x-ray examination of this case disclosed a definite patch of consolidated lung conforming to that outlined by the physical signs. It was not clear, however, whether there was in addition some fluid encapsulated in the interlobar fissure.

In this case it seems apparent that the original trouble was an influenzal bronchitis, and just as this was subsiding a small pneumonic patch developed which was responsible for the continuance of the fever.

CASE II.—This man states that his illness began three days before his admission to the hospital with malaise, anorexia, and a drowsy feeling. When we first saw him he complained of pains all over the body and sore throat. He had a cough and some mucopurulent expectoration. Physically he was a poor specimen, being anemic and under weight. The tongue was heavily coated. The blood-pressure was 120 systolic and 75 diastolic. The physical signs on admission were those of a bilateral bronchitis. There were no localizing signs.

As you will see from the temperature chart (Fig. 133) he had a high fever at the time of admission (104.4° F.). In the course of three days this had fallen to normal and remained so for one day, when it abruptly rose again. At this time he also developed marked nervous symptoms chiefly in the form of constant twitching and a muttering delirium.

Examination of the chest at this time showed that the râles had a sharp, consonating quality, especially at the right base; otherwise there were no signs of consolidation. Two days later, however, an area of flatness on percussion, distant bronchial breathing, and bronchophony were clearly made out near the angle of the right scapula. A blood count at this time showed 3,750,000 red cells, 19,040 white cells, and 75 per cent. hemoglobin.

This second case illustrates very well the danger of considering the attack over when the temperature reaches normal and remains so for a few days. No patient can be considered free from the danger of a serious complication, such as bronchopneumonia, until at least seven or eight days have elapsed without fever. We have had a number of patients admitted to the ward with bronchopneumonia or lobar pneumonia who stated that they had had fever for a few days and then gotten up, and in some instances returned to work.

Neither of these cases offered any great difficulty in making a diagnosis, as the signs of consolidation were evident. But even if most of the signs, commonly attributed to consolidation of the lung, are absent, one should rarely fail to make a diagnosis. In many instances a diagnosis can be made even in the absence of conclusive physical signs.

In the first place we have come to learn both from autopsy and  $x$ -ray studies that some evidence of bronchopneumonia is present in the large majority of cases. Therefore if the fever is persistent, the respiratory rate high, and the patient toxic we can be almost certain that such a condition exists.

As to the physical signs, there are two which are worthy of special emphasis: (1) Bronchophony. In case after case it has been possible to localize a small patch of consolidation by the exaggeration of the voice sounds at a given point. This will happen when bronchial breathing cannot be heard and there is no percussion change. (2) The sharp, consonating character of the râles. In no other condition with which I am familiar are the râles as sharp and metallic in quality, and this feature will often antedate for a number of days definite signs of consolidation, and may be the only tangible evidence of the condition which we can obtain.

The location of bronchopneumonic areas is usually about the angle of the scapulæ. Our experience with the  $x$ -ray examination of these cases has shown that the bronchopneumonic areas are almost invariably of greater extent than is shown by the physical signs.

Another interesting feature of these influenzal broncho-

pneumonias is the persistence of the consolidated areas. Not only is the febrile attack often unduly prolonged, but, in addition, even when the constitutional signs have disappeared, both the physical and x-ray examinations will show that there is very little clearing up of the lesions. This is to be ascribed, I believe, to the fact that influenzal pneumonias are so often characterized by inflammatory change in the interstitial tissues of the lung. There is, therefore, not only the exudate to be gotten rid of, but, in addition, the interstitial changes.

A more remote change is bronchiectasis. I have already alluded to the fact that in many cases the bronchial wall is distinctly swollen and softened. This fact, in conjunction with the relatively long-standing pulmonary changes, is probably a very potent factor in bringing about a permanent distention of the bronchi. This may explain also the fact that the one organism most constantly associated with bronchiectasis is the influenza bacillus.

#### TUBERCULOSIS AND INFLUENZA

From what we have already seen of this epidemic it is plainly evident that we are going to experience considerable difficulty in the next few months in regard to tuberculosis. The mistakes are going to be of two kinds. On the one hand, persistent and slowly resolving patches of influenza pneumonia, especially if located at the apex of the lung, are going to be mistaken for tuberculosis. On the other hand, known cases of tuberculosis which have undergone an attack of influenza are going to offer considerable difficulty in determining whether the increase in the physical signs is due to an extension of the tuberculous process, or whether it is a slowly resolving influenza pneumonia which will gradually clear up. The following case illustrates the first-named difficulty:

This patient had a persistent small patch of consolidation which was apparently responsible for a continuance of the fever and respiratory symptoms. He came into the ward originally because of epigastric pain from which he had been suffering for several weeks. Nine days after his entrance into the

ward and at a time other patients suffering from influenza had been admitted he developed malaise, headache, cough, rusty sputum, fever, and some dyspnea. Examination of his chest showed the signs of a diffuse bronchitis; a few days later a definite

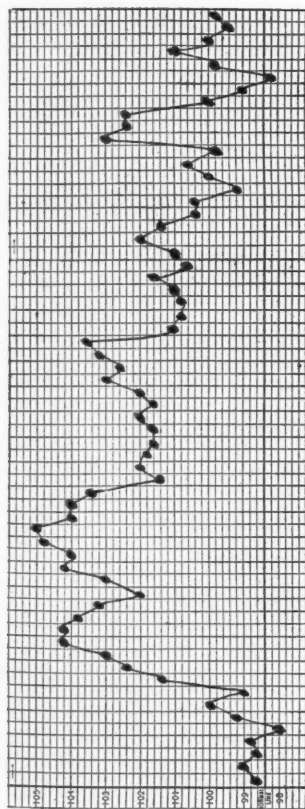


Fig. 134.—Influenza, with possible active tuberculosis following.

patch of pneumonic consolidation was made out about the angle of the left scapula.

After five or six days the temperature declined to 100° F. (Fig. 134) and kept at this level. In addition, there was a marked

increase in the amount of sputum, which was mucopurulent in character.

In view of the fact that his history showed the occurrence, eleven years before, of an illness in which the chief symptoms were cough and night-sweats, it was thought that the persistence of the fever might be due to a latent tuberculosis which had become active as the result of the influenzal attack.

Examination of his chest showed that the percussion note at the right apex was impaired, the breath sounds were rough in character, and the vocal resonance distinctly exaggerated. The sputum, however, was negative for tubercle bacilli.

The x-ray examination showed the presence of a sharply defined area, about the size of a silver dollar, at the level of the second rib on the right side. There was nothing in the appearance of the picture which suggested tuberculosis.

This case illustrates an important point because of the location of the consolidated area. The physical signs of a small area of consolidation or infiltration at the apex of the lung is highly suggestive of tuberculosis. Indeed, under ordinary conditions such a lesion is almost certainly tuberculous in character. During the presence of an epidemic of influenza, however, it must be kept in mind that a small patch of influenzal pneumonia may occur in and be limited to the apex. During such a time, therefore, the occurrence of fine crackling râles limited to the apex are to be looked upon as possibly tuberculous in origin, but not necessarily so.

Before the present epidemic had become generally recognized a young Italian girl came to the Phipps Institute Dispensary complaining of cough, expectoration, and malaise. Examination of her lungs showed the presence of fine crackling râles localized in the right apex. As she had slight fever the diagnosis of tuberculosis seemed more than probable. A few days later, however, she returned to the Dispensary because of general pains, and at this time fine sticky râles were heard all over both lungs. It was evident, therefore, that she had influenza and not tuberculosis.

An error in this direction is, however, not so apt to occur as

when the acute signs of the influenza have disappeared and there is left behind a small pneumonic patch at one apex which is slow in resolving. Under such circumstances it will be the part of wisdom for the duration of this epidemic, and for some months to come, to suspend judgment in such cases.

A conservative attitude must also be maintained in regard to patients known to be tuberculous and who have passed through an influenzal attack. Already I have seen several instances of this kind. It is to be borne in mind that a lung which has been infected with tuberculosis, even if the process has been arrested for some time, is extremely susceptible to acute respiratory infections, and because of the fibrous tissue and other changes about the lesion such infections are apt to persist for a much longer time than in a normal healthy lung. In the cases which have come to my notice it has not been possible to state at once whether the process is due to a lighting up of the old tuberculous lesion or whether the râles are due to the influenza attack and will eventually disappear, or, if râles have been present previously, that they will diminish.

It is the better judgment in such cases to withhold an immediate opinion. The patient should be seen at intervals, and by the end of six weeks or so it can be pretty definitely determined which condition we have to deal with, because by that time both the physical signs and the symptoms will make it apparent as to whether there has been a lighting up or an extension of the tuberculous process.

#### **TUBERCULOUS PNEUMONIA**

The next cases also introduce the subject of tuberculosis, but in a different form.

CASE I.—The patient was a male, aged twenty-two years, a tailor by occupation, who was admitted to the hospital October 13th. He gave as his chief complaints cough, pain in the chest, and jaundice, the latter having appeared the day before. He stated that he had been ill for two weeks and attributed it to working overtime for a considerable period.

His illness began with headache, slight sore throat, and cough.

The expectoration was quite profuse and of a thick, greenish, mucopurulent character. It did not show any evidence of blood. He denied the occurrence of any serious illness in the past and there was nothing of importance in his family history.

*Examination.*—The patient was very weak, had evidently lost considerable weight, and was markedly anemic in addition to being jaundiced. The tongue was thickly coated with a blackish looking fur. The tonsils were enlarged and the throat was congested.

Expansion of the left lung was restricted. The percussion note was markedly impaired all over the left chest. On auscultation fine sticky râles, bronchial breathing, and marked exaggeration of the voice sounds were heard over the left side.

The right lung did not show any abnormality.

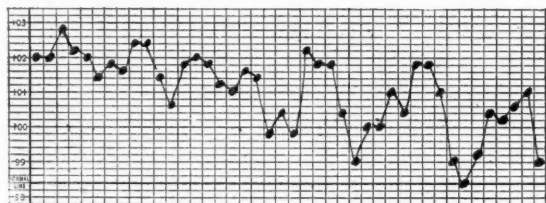


Fig. 135.—Fever chart from case of tuberculous pneumonia.

Aside from a very rapid pulse-rate the heart showed nothing abnormal.

Five days later (October 18th) the same signs were present over the left side. At this time it was noted that the temperature was becoming more remittent in type (Fig. 135). Owing to the fluctuating character of the temperature, the anemia, the emaciation, the character of the sputum, and the absence of severe respiratory distress and toxemia, so commonly seen in lobar pneumonia, a tuberculous origin of his trouble was suspected. Examination of the sputum showed the presence of large numbers of tubercle bacilli. (The patient died suddenly on the tenth day of his stay in the hospital.)

**CASE II.**—A male, aged twenty-one, was admitted to the hospital complaining of cough, headache, and pain in the limbs.

He is positive in his statement that up to four weeks ago he had been as always—perfectly well. At this time he developed a headache, felt drowsy, and had some cough and expectoration; the latter symptoms he attributed to a “cold.” Although he felt badly he kept up and about, but finally had to take to his bed; a few days’ rest made him feel much better, but he still complained.

He denies having had a cough prior to the onset of his present attack four weeks ago. He raises a considerable quantity of mucopurulent material; this became blood-streaked five days prior to his admission and has become more and more noticeable.

He had had no night-sweats. Since the beginning of his illness, four weeks ago, he has lost about 15 pounds.

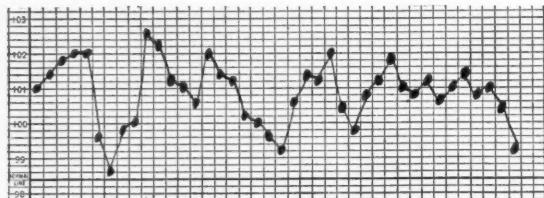


Fig. 136.—Fever chart from case of tuberculous pneumonia.

The family history is negative aside from the fact that his mother died at the age of twenty-six from causes unknown to him.

Aside from an indefinite history of rheumatism at the age of fourteen he has never been ill.

His symptoms on admission were quite characteristic of influenza. Nor was the blood-streaked sputum or the preponderance of physical signs on the left side incompatible with such a diagnosis. He had, however, a considerable amount of fever (Fig. 136), the persistence of which, in view of the length of his illness, was suggestive of something more than simple influenza or influenza complicated by pneumonia.

*Examination.*—A more careful examination of his chest showed some flattening beneath the left clavicle and distinct

limitation of motion all over the left chest, especially at the apex. The percussion note was markedly impaired over the upper part of the left lung and slightly so over the remainder of that side. The breath sounds were bronchovesicular in type and somewhat obscured by numerous crackling and subcrepitant râles. Aside from some exaggeration of the whispered voice at the left apex, there were no definite signs of cavity.

The heart showed nothing abnormal.

The sputum was found to contain numerous tubercle bacilli.

As in the previous case, this patient did not present the toxic appearance so commonly seen in the influenza pneumonia cases.

The x-ray examination showed an extensive tuberculous infiltration of the left upper lobe and the presence of two small, deep-seated cavities. The left lower lobe was the site of a diffuse, hazy, infiltrated area quite characteristic of the area of consolidation seen in influenza cases. This will, in all probability, eventually disappear. The right lung shows some scattered patches which are probably influenzal in origin.

The prognosis in this case is bad, although at present it is problematic how much of the trouble on the left side is tuberculous and how much is due to an influenza infection. It is quite possible that the signs of bronchitis over the lower part of the left lung are due to influenza and will eventually clear up.

(*Note*—six weeks later. The patient is failing steadily. Temperature hectic in type. A second x-ray examination shows pretty conclusively that the entire process in the left lung is tuberculous, and, in addition, there are scattered foci about the hilus of the right lung.)

Both of these patients, especially the second one, insisted that their health had been unimpaired up to the onset of their present illness. This may or may not be true. It is hard to believe that such a wide-spread process, as indicated by the physical signs, could have progressed to the present point without some manifestation of ill health. While it is quite true that similar cases are frequently seen, it is usually possible on questioning them closely to elicit the information that they have felt below par for some time without, however, their being able to indicate

anything more definite than that they felt "run down" and out of sorts.

These cases illustrate very well how closely an acute and widespread tuberculous process may simulate lobar pneumonia. There is no commoner mistake in the diagnosis of chest conditions. In many cases there is nothing to suggest that the acute symptoms—such as a high and continuous fever, pain in the side and blood-streaked sputum plus the physical signs of consolidation—are due to anything else than a lobar pneumonia. The first suspicion that the condition may, in reality, be tuberculous is apt to occur when the process is unduly prolonged and the temperature begins to fluctuate widely.

I have never seen tuberculosis of the lungs which seemed to develop as the result of an attack of croupous pneumonia. For some reason tuberculous individuals, with an active lesion, seem to bear an attack of pneumonia remarkably well. On the other hand, it is a common experience to have patients, and often physicians, state that the tuberculosis followed or was caused by an attack of pneumonia. This is rarely the case. It is to be borne in mind that tuberculosis is not invariably insidious in its onset. It may begin acutely, and when the patient is first seen there may be a considerable area of consolidation at the summit of one or the other lung. In addition, the symptoms may be those commonly encountered in pneumonia. This acute attack may subside and the case remain latent for a long time; or it may become subacute and gradually merge into the familiar chronic ulcerative type of the disease; or, finally, as in the case of one of the patients under discussion, the process may progress rapidly to a fatal termination.

How are these cases to be detected? In the first place it is to be remembered that if the process is limited to or more marked in the lower lobes, it is most certainly not tuberculous. If an upper lobe is involved and the base is clear, the lesion may or may not be tuberculous. In such cases tuberculosis is to be thought of when the fever is unduly prolonged, and especially if after a week or ten days it becomes remittent or intermittent in character. Furthermore, there is usually marked loss in

weight, sweating is common, and considerable prostration is present. Additional evidence in favor of tuberculosis are a history of that infection in the family; a more or less prolonged period of ill health prior to the onset of the acute symptoms; and the occurrence in the past of an illness which is suggestive of tuberculosis, as, for instance, a hemoptysis, an attack of pleurisy, or the occurrence of a fistula in ano. These symptoms are often spoken of as prodromal and may precede definite pulmonary manifestations by months or even years.

Since the start of this epidemic I have already seen a number of tuberculous patients who had had influenza. In nearly every instance it was impossible to tell from the physical signs whether the extension of the trouble was tuberculous in nature or whether it was due to the influenzal infection. Even an x-ray examination does not enable one to form a definite opinion.

#### POSTINFLUENZAL PLEURAL EFFUSION

We have had a number of pleural effusions developing during or after the subsidence of an influenzal pneumonia. Effusions developing during the course of an attack of pneumonia unless massive are commonly overlooked during the acute stage of the primary trouble. An effusion developing under these circumstances is commonly mistaken for a persistent or unresolved pneumonia. When you hear of a pneumonia being unduly protracted and are told that the physical signs persist, a pleural effusion is one of the first things to think of. In my experience the great majority of so-called unresolved pneumonias prove to be a pleural effusion, free or encysted or a tuberculous process. The one exception I would make to this rule is a condition so frequently seen in influenza epidemics. During such an epidemic we not infrequently encounter individuals in which a patch of consolidation will persist for some weeks.

The 3 cases which I wish to discuss with you now are of interest not only because they are excellent examples of an effusion developing after or during an influenza pneumonia, but more particularly because of the physical signs.

CASE I.—The patient was admitted complaining of cough and pain in the stomach.

He had been ill for one week. The attack began with a chill, followed by headache, pain in the stomach, cough, and the expectoration of a thick yellow sputum. For the first few days of his illness he vomited constantly.

On admission it was noted that his throat was congested and that the tonsils were enlarged and also congested.

Examination of the chest showed increased fremitus, marked impairment of the percussion note, bronchial breathing, bronchophony, and fine sticky râles over the left lower lobe posteriorly. There was a systolic murmur at the apex, but no cardiac enlargement. The blood showed 4,300,000 red cells, 4600 white cells, and 75 per cent. hemoglobin. The urine contained a few hyaline casts. At a later examination a few sticky râles were heard at the right base posteriorly.

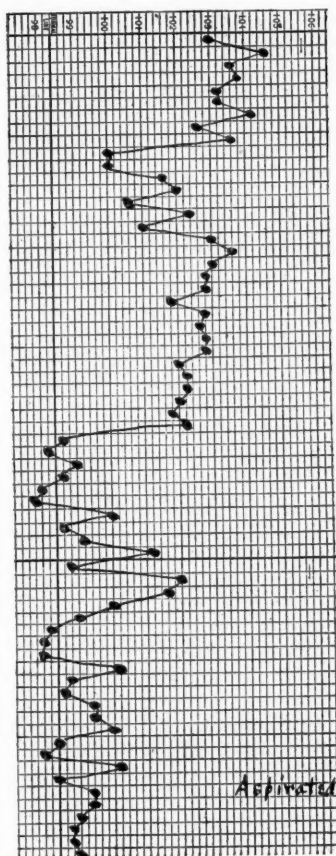
The seventh or eighth day of his stay in the hospital the temperature dropped abruptly to normal and remained so for a day, when it rose again and continued irregularly (Fig. 137). Owing to the recurrence of the fever we became suspicious that some additional trouble had occurred.

On examining his chest it was found that the expansile movements over the lower half of the left chest were absent and the apex-beat of the heart could not be seen. Tactile fremitus was absent and the percussion note was flat. On auscultation, loud bronchial breathing was heard all over the flat area which practically coincided with the position of the lower lobe of the left lung. In addition, there was marked bronchophony and whispering pectoriloquy. The bronchophony, however, had a nasal quality (egophony), especially over the upper limits of the dull area.

In spite of the auscultatory findings a diagnosis of effusion was made for the following reasons: The almost complete lack of expansion; the absence of tactile fremitus; the flat percussion note, and the fact that the heart was displaced to the right as shown by the absence of the apex-beat from its normal position, its location by auscultation near the ensiform cartilage, and the

extension of the right border beyond the edge of the sternum. An exploratory puncture proved the correctness of the diagnosis.

Fig. 137.—Effusion following lobar pneumonia. Often mistaken for "unresolved pneumonia."



The fluid first removed was reddish and turbid in appearance. A few days later 680 c.c. of purulent fluid were removed.

CASE II.—In this case the history is much the same. The patient was admitted to the hospital complaining of cough and pain in the side.

He stated that he had been ill for twelve days and that the attack had started with a drowsy feeling, headache, pain in the back and limbs, and nausea. Three days before his admission he had a chill followed by fever, cough, bloody expectoration, and pain in the side.

On admission he had sordes on the tongue and lips, a heavy whitish fur on the tongue, and the pharynx and tonsils were reddened and congested.

There were numerous fine crackling râles in both lungs. Over the left base posteriorly the percussion note was dull, and bronchial breathing and bronchophony were heard. Four days later it was noted that the bronchophony and the bronchial breathing were distant, and anteriorly below the level of the left nipple coarse râles were heard. It is possible that the effusion was beginning at this time, because five days later the percussion note was flat, the bronchial breathing louder, and the bronchophony had a distinct nasal quality.

The heart, which was previously noted to be in its normal position, was displaced to the right.

An exploratory puncture showed the presence of blood-tinged, turbid fluid.

The physical examination of this patient gave practically the same findings as in Case I, the bronchial breathing, the bronchophony, and whispering pectoriloquy being markedly exaggerated. As in the previous case, an exploratory puncture showed the presence of a reddish-yellow, cloudy fluid.

CASE III.—In this instance the patient was admitted after an illness of some eight or nine days' duration. His case differed from the previous two in the fact that the effusion developed coincidently with the pneumonia.

The patient stated that he had been ill for seven days, the attack beginning with chilly sensations, high fever, dizziness, and cough. At the time of his admission he was expectorating a considerable quantity of mucopurulent sputum which later became blood tinged.

On admission the patient was very nervous and slightly dyspneic.

The physical examination revealed the same physical signs as in the two previous cases. There was this slight difference, however, the signs were limited to the lower third instead of the lower half of the left chest, and, in addition, were not marked anteriorly. This, I think, can be attributed to the fact that he was examined while lying on his back and on his right side, thus allowing the small effusion to gravitate away from the front. In large effusions, with the consequent crowding together of the thoracic contents, the gravitating effects are not nearly so well marked as in the case of small collections of fluid.

The apex-beat was not seen, but was located behind the sternum; the right border of the heart extended well beyond the right border of the sternum.

The blood count showed 4,180,000 red cells, 6000 whites, and 80 per cent. hemoglobin. The urine contained a trace of albumin and numerous granular casts.

Fluid was suspected because of the egophony and the displacement of the heart. This was confirmed by an exploratory puncture which showed the presence of a bloody, turbid fluid.

On the third day of his stay in the hospital and the tenth day of his illness the temperature fell to normal by crisis.

How then are we to distinguish between a pleural effusion and a consolidation of the lung? In both conditions the expansion of the lung is interfered with; in both the percussion note is dull or absolutely flat; tactile fremitus should be exaggerated over a consolidation and absent over fluid, but it varies greatly, and, therefore, does not furnish sufficiently strong evidence one way or the other. Bronchial breathing and exaggeration of both the spoken and whispered voice sounds may be present over fluid as well as over consolidated lung tissue, although it is the exception and not the rule. The cardinal factor is displacement of the viscera. Thus, if the effusion is on the left side, the heart is crowded to the right, and the stomach, by reason of pressure on the diaphragm, is pushed downward, thus obliterating Traube's semilunar space. This space normally gives a tympanitic note due to the portion of the stomach which lies between the left lower margin of the lung, the right border of the spleen,

and the left border of the liver. It is a sign of relatively little value in small effusions because the weight is insufficient to displace the diaphragm downward. In massive effusions, especially if purulent, the space is usually obliterated.

If the effusion is right sided the apex-beat of the heart is displaced to the left, and if the collection of fluid is large the liver is displaced downward.

The position of the heart is really the most important consideration in these cases, and the fact that it is displaced to one side or the other can usually be determined by inspection. In thick-chested individuals it may be necessary to locate the position of the apex by means of auscultation. The point at which the heart sounds are heard with the greatest distinctness can be taken for the position of the displaced apex.

It is well recognized that in the great majority of pleural effusions there is silence over the area occupied by the fluid. Either the breath and voice sounds are feeble and distant or entirely absent. How are we to explain the fact that in some instances of pleural effusion there is a distinct transmission of both the breath and voice sounds?

Until a few years ago there was no adequate explanation. A number of hypotheses were advanced, but none of them were satisfactory.

As the result of the work of Montgomery and Eckhardt on pulmonary acoustic phenomena (Tenth Annual Report of the Henry Phipps Institute, 1915) the explanation is clear. In their study of the physics of sounds developed within the thorax they concluded that the most important factor is diffusion. The normal lung, for instance, transmits sounds produced in the larynx and carried along the trachea and bronchi very poorly because the parenchyma of the lung allows of diffusion. The result is that there is a distinct modification of the sound as it reaches the ear at the chest wall. The same is true of partially relaxed lung tissue. On the other hand, if the lung is consolidated or tightly compressed there is little or no diffusion. The application of this in cases of pleural effusion is as follows: If the lung is only slightly compressed by the effusion the bron-

chial qualities of the breath and voice sounds as they exist in the trachea are widely diffused on striking the relaxed lung, and are nearly or completely lost in passing through the fluid. Hence over the ordinary effusion there is silence. On the other hand, if the lung is tightly compressed or, as in the case of pneumonia, is still in a state of complete or partial consolidation, there is no diffusion of the tracheal sounds as they pass through the lung and, in turn, through the fluid. As a result of this lack of diffusion the bronchial quality of the breath and voice sounds as they occur in the trachea are directly transmitted to the chest wall.

#### LOBAR PNEUMONIA WITH RELAPSE

In addition to bronchopneumonia there have been many examples of the lobar type of the disease in the wards. Many of these cases, however, were atypical in their clinical course. The fever curve often showed distinct departures from that usually associated with frank croupous pneumonia. Thus, instead of pursuing a continuous course with slight intermissions, the fever was commonly irregular and rarely dropped abruptly by crisis. Usually the fever declined by rapid lysis. Another characteristic of the croupous type of the disease is the viscid, tenacious sputum. Indeed, this may be said to be almost pathognomonic of croupous pneumonia. In these influenza pneumonias the sputum was rarely viscid. As a rule it was mucopurulent and frequently blood streaked, but the blood was not intimately mixed with the sputum, as is the case with the rusty sputum. In addition, small hemoptyses were not infrequent. These variations are probably to be ascribed to the fact that a pure pneumococcus infection was rarely present. In addition to the pneumococcus, the influenza bacillus, the streptococcus, and other organisms also were present. Furthermore, the gross appearance of lungs the site of lobar involvement often suggested that the process was primarily a number of foci of bronchopneumonia, and that these coalesced.

It also seems to have been a common experience that the left lower lobe was the part most affected instead of the right lower lobe. In the wards of the University Hospital involvement of

the left lobe occurred in about 70 per cent. of the cases; my experience at Emergency Hospital No. 2 was similar to this.

The case I wish now to show you is an excellent illustration of a relatively rare condition, namely, a relapse. Although there is some confusion as to what constitutes a relapse, it may be said that "if a lung, after ordinary croupous pneumonia involving one or several lobes, becomes normal after the fever has terminated by crisis or lysis, the patient is convalescent; and if at least three days or several weeks after the defervescence a new infiltration of the same or other lobes with all the characteristic phenomena of a local and general routine occur, a relapse has, without question, taken place" (Wagner). A relapse is noted in about  $\frac{1}{2}$  of 1 per cent. of cases.

CASE I.—Two weeks before his admission to the hospital the patient was apparently taken with influenza. He had a cough, yellowish expectoration, headache, and pain in the back. Three days before his admission he got up, but at once felt badly, and was shortly afterward seized with a chill, high fever, a return of the pain in his back, and an increase in the cough.

On his admission it was noted that the pharynx and tonsils were markedly congested and the tonsils were also enlarged. Examination of the blood showed 4,460,000 red cells, 5920 white cells, and 80 per cent. hemoglobin. The urine contained a few hyaline casts.

Posteriorly over the right lower lobe the percussion note was dull, the breathing bronchial in character, and the voice sounds markedly exaggerated. A few coarse râles were heard at the left base.

The temperature fell irregularly and remained normal or nearly so for four days, when it abruptly rose again (Fig. 138). This was accompanied by an increase in the respiratory rate, the cough became worse, and the amount of sputum increased.

Two days after the second febrile period developed examination of the chest showed the classical signs of consolidation over the left lower lobe. The second attack lasted for nine days, when the temperature fell by rapid lysis to normal.

The one feature that has not been affected is his pulse-rate.

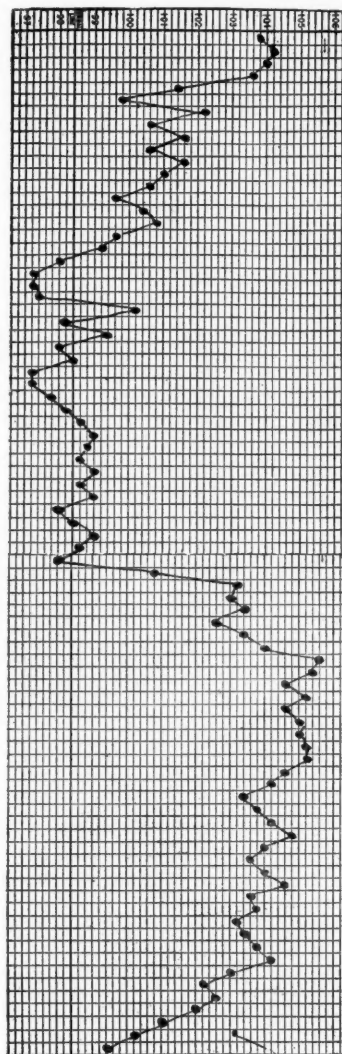


Fig. 138.—Chart showing relapse in pneumonia.

This continues unduly high and, in addition, the heart sounds lack good muscle tone. This is probably due to a slight degree of acute myocarditis. In practically all of the acute infections some degree of acute myocarditis occurs, although, as a rule, it is not demonstrable clinically. In this case the two attacks occurring so close together may have involved the heart muscle more than ordinarily.

CONTRIBUTION BY DR. JOHN B. DEAVER

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## THE SURGICAL COMPLICATIONS AND SEQUELÆ OF INFLUENZA

INFLUENZA, like the poor, we have, and probably shall have, with us always. Fortunately, very severe epidemics occur at rather widely separated periods of time, and more fortunately few are so virulent and so general as the one that is now on the wane. George B. Wood, in his *Practice of Medicine* (5th ed., 1858), described an outbreak in Rome in 1850, during which 9000 persons are said to have died of the disease.

Since there is scarcely any part of the bodily organism that is spared some immediate or remote effect of this insidious disease, it is only natural that some of its complications and sequelæ should be of surgical importance and interest, more especially so since any latent infection is apt to flare up during an attack of influenza or during the period of lowered resistance that follows. We have lately had several cases that have recently recovered from influenza who came to operation for intestinal obstruction. The intestines were knotted and bound down by plastic exudate and adhesions resembling a tubercular peritonitis. The impression was gained that the condition may have been the direct result of the influenza, but there was no positive evidence of the fact. A number of authors have noticed an increased incidence of appendicitis in influenza years. Many of such cases most likely are so-called pseudo-appendicitis due to the gastrointestinal symptoms that so often are part of the syndrome of influenza, and others are undoubtedly genuine appendicitis resulting from the increased virulence of the usual intestinal micro-organisms, and particularly to a predisposition on the part of the patient to appendix trouble. This explanation—lowered resistance and diathesis—undoubtedly also serves for the various neu-

ralgias, diseases of the bones and joints, as well as for phlebitis, parotitis, and other more or less unusual conditions that may follow influenza and require surgical intervention.

The term "surgical complications and sequelæ of influenza" is more apt to imply the effects of the pulmonary involvement which so frequently forms part of the picture and which is so prominent a feature of the present epidemic.

The medical aspect of the subject, especially the recognition of the presence of pus in the thoracic cavity, is of the utmost importance to the surgeon. Were we always sure of the physician's ability to make an early diagnosis of empyema, the surgeon's task would be considerably lightened, the patient's period of convalescence considerably shortened, and, most important of all, there would be much less, if any, risk of such undesirable results as displacement of the heart, deformities of the chest, and impairment of the function of the lung that may follow the more extensive operation required when intervention is long delayed.

Aspiration under proper aseptic precautions is a comparatively simple and safe procedure; oftentimes, however, owing to improper technic no liquid is obtained, and the physician being satisfied that none is present (when perhaps the pleura has not even been entered), the case goes on to chronicity. In early cases, especially in children, the fluid is apt to be serous, but even so, it should be withdrawn. When the fluid is sero-purulent or purulent withdrawal by aspiration is desirable as a preliminary step to thoracotomy or rib resection. Aspiration is of the utmost value in allowing the lung to expand and the displaced heart to recover its position. The relief afforded also puts the patient in better condition for, and lessens the danger of, subsequent operation, especially if a general anesthetic is to be used.

In addition to frequent needling and the information derived from physical signs the fluroscope and x-ray are, of course, most valuable aids in diagnosis, in locating the collection and possible encapsulation of pus, and in indicating the most favorable point for drainage.

A rational operation for empyema is the one devised by Lilienthal, in which a wide opening in the thoracic cavity is obtained by means of a long costal incision and wide rib-spreaders. This gives ample exposure and permits the breaking up of adhesions and the removal of pyogenic membranes and allows full expansion of the lung. The wound is closed completely except for a wick of rubber tissue at each end of the incision.

In the streptococcic pleuritis observed in the extensive epidemics of pneumonia occurring during the past year at the various military training camps, it seems that late operation gave better results than early intervention. Unlike the pleuritis following ordinary lobar pneumonia the effusion in the streptococcic cases appears early; in fact, often is the first sign of infection of the respiratory tract, and operation in the acute stage, in addition to other risks, presents the danger of collapse of the lung from pneumothorax as well as a possible infection of the blood-stream from absorption of the streptococci from the fresh surfaces of the wound.

A valuable suggestion is that all pneumonia cases at the end of the second week be subjected to x-ray examination for early detection of any fluid that may be present and which cannot always be detected by physical signs, as, for example, an isolated abscess cavity, or when the pus is confined within a fibrous exudate, or when a slow formation of pus plasters itself over the surface of the lung in a thick layer through which the sounds of the consolidated lung are transmitted and the presence of the fluid remains undetected.

Dr. Pancoast has called my attention to the fact that the effusion in influenzal empyemas seems to show a tendency to become plastic very quickly and to wall off, so that in the majority of cases it is localized. This was found to be the case in 16 out of 25 empyemas observed at the University Hospital. As a result of this plastic exudate the pleura, even though the effusion be quickly absorbed, becomes markedly thickened, and the resulting adhesions cause crippling of the lungs.

In 35 cases treated by us at the University Hospital and at the Lankenau Hospital, 13 were interlobar empyemas, several

of which had ruptured into the lung, and in one instance pus from the general cavity had broken through into the lung. Thoracotomy except in rare circumstances is decidedly not the proper procedure for these cases, in fact, is mentioned here only to be condemned.

The operation of choice for empyema is rib resection—opening the pleural cavity and exploration with the gloved finger or the hand, thus effectively reaching all pockets of pus; flushing and wiping the cavity with Dakin's solution, and providing continuous and free drainage until the fluid returned is practically sterile. McKenna, reporting the work of the empyema team at Camp Pike, advocates the drainage of the pleural cavity in all cases irrespective of the character of the pus. He uses a No. 14 French rubber catheter and introduces it by means of a trocar and cannula just large enough to thread the catheter into the pleural cavity, connects the catheter with a 100-c. c. glass syringe, and aspirates. If the pus is too thick for aspiration a small amount of Dakin's solution is run in in order to liquefy the pus; by repeating the procedure the entire pleural cavity can be evacuated. This may be very well, but to my mind it is prolonging the agony.

The closure of the wound is a matter of circumspection unless there is a free expansion of the lung. Failure of the latter to expand is probably an indication of some latent process which may result in a recurrence of the empyema, or some deformity and impairment of function.

In 2 cases I closed the wound at once, and both have done well. This immediate closure of the wound when it can be done is useful in overcoming a possible pneumothorax, by preventing the entrance of air from without. Delbert, in a recent address before the College of Physicians, also advocated this procedure and suggested that if necessary it is an easy matter to open the suture and insert a tube.

Operation in our cases was usually done under nitrous oxid anesthesia, and consisted of resection of about 2 inches of rib, the sixth, seventh or eighth, according to indications, evacuation of the pus, wiping the cavity, and continuous drainage with

gauze or rubber, only occasionally using Carrel tubes. Faithful and intelligent dressing daily with Dakin's solution has given excellent results in our experience. We have sometimes found it advisable to discontinue the use of Dakin's solution after about ten days and to substitute carbolic, permanganate, or saline solution.

When necessary a more extensive opening is made in order to explore the thoracic cavity, break up adhesions, or search for an obscure source of pus.

One of our patients, now convalescing at the Lankenau Hospital, on the ninth day after an abdominal operation developed chills, fever, and indications of right lobar pneumonia. He coughed up about 30 c.c. of thick, bloody pus, which on examination was found to contain pneumococci and influenza bacilli. At operation one week later, after locating the pus with the aspirating needle, a vertical incision was made over the seventh rib, the periosteum removed and the rib exposed, and 2 inches of the latter resected. The pleura was then opened and 1000 c.c. of cloudy, yellow fluid withdrawn. The cavity was washed out, rubber tubes were inserted for drainage, and the wound closed. The relief following the operation was only temporary. At a second intervention, fifteen days after the first, the operative incision was enlarged, portions of the sixth and eighth ribs removed, and the thoracic cavity explored. No pus was obtained. The lower lobe of the right lung was found to be collapsed and rather hard to palpation. It was adherent to the diaphragm, but was functioning to some extent. The thoracic cavity was washed out with Dakin's solution, and the wound closed, one piece of gauze left in for drainage. The wound cavity is now being dressed daily with Dakin's solution, and the patient is making satisfactory though slow progress toward recovery. This is one of the cases of interlobar abscess rupturing into the lung, to which reference has already been made.

During the present epidemic we have treated 35 cases of influenzal empyema, with a mortality of 11.6 per cent. (University Hospital, 25 cases, 2 deaths; Lankenau Hospital, 10 cases, 2 deaths.)

In 24 cases the empyema was localized:	15 right-sided	} 13 interlobar
	7 left-sided	
	2 double	
In 10 cases the empyema was diffuse:	6 right-sided	
	3 left-sided	
	1 diffuse left, localized right	
The series included only	1 empyema necessitans	

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Total, 35

## BACTERIOLOGIC STUDY OF SPUTUM IN THE RECENT EPIDEMIC

**Material for Study.**—This consisted of sputum from about 80 cases of the disease in which bronchopneumonia was diagnosed by the clinician. Sputum was obtained in sterile bottles, brought to the laboratory as soon as collected, and cultures made after washing; spreads were made and stained with methylene-blue and also by Gram's method. The sputa were washed in two or three changes of sterile salt solution before inoculation upon the proper culture-media. In addition to the sputum, studies were also made from swabs and cultures from the nasopharynx and throats of these individuals. The spreads were treated in exactly the same manner as were those of sputum.

**Culture-media.**—The culture-media used were blood-agar and blood-smear agar. The blood-agar consisted of 2 parts of agar and 1 part of blood added together at 42° to 45° C., mixed, slanted, and kept in the incubator at least twenty-four hours to insure sterility. The blood-smear agar was made with human blood obtained from the finger, under sterile precautions, smeared heavily over the surface of the agar, and incubated for twenty-four hours to insure sterility before using. In addition to the sputum and throat spreads a few inoculations were made from the fluid which exuded from the throat and nose and sometimes the ears of those dead of the disease.

**Results.**—In the spreads of sputum the most conspicuous organism was a coccus which corresponded morphologically and in staining reaction to the pneumococcus. No attempt was made to determine the type of the pneumococcus. The next most abundant organism was the *Micrococcus catarrhalis*, which occurred in very large numbers; and then a streptococcus.

Besides these three prominent organisms we also encountered the bacillus of Friedländer occasionally; pseudodiphtheria bacilli were common and staphylococci were very noticeable. In only six or seven specimens, after a very long search, could a bacillus which corresponded in morphology and staining reaction to the *Bacillus influenzae* be demonstrated. No one familiar with the classical picture of the *Bacillus influenzae* could mistake it, for the simple reason that this organism is distinct as a short, thin, straight, Gram-negative bacillus occurring in clumps and usually found within the leukocytes. Filamentous forms are not common unless the organism is cultivated upon a medium suitable for it and in which involution forms occur in the very early hours of its growth.

**Physical Characteristics of the Sputum.**—In some of the specimens the sputum was of a bright reddish-pink color, and in others the sputum was of the ordinary yellowish or grayish-white color in which "chunks" of this reddish-pink material were present. In still others the sputum resembled the characteristic rusty sputum of pneumonia, while the majority were grayish or yellowish-gray in color with no special characteristics. In a number of spreads of specimens of sputum spiral organisms were present of variable sizes, and which no doubt represented the *Spirochæta microdentia* and *Spirochæta macrodentia* and the spirillum of Vincent. These organisms were present *only* in spreads, and were undoubtedly the result of defective teeth or of mouths which had not been properly cleansed before the collection of the specimen for study. It is my belief that their presence was of no etiologic significance.

**Studies of the Throat Spreads and Cultures.**—In the majority of the spreads almost the same organisms and in the same order of prominence were encountered as in the spreads of sputum. It might be mentioned that the *Micrococcus catarrhalis* was found in larger proportion in the throat spreads than in the sputum. The cultures showed a variety of organisms; in very few a hemolytic streptococcus was observed, and in the majority of cases staphylococci were especially common. Pneumococci, pseudodiphtheria bacilli, and in 4 or 5 cases the bacillus of

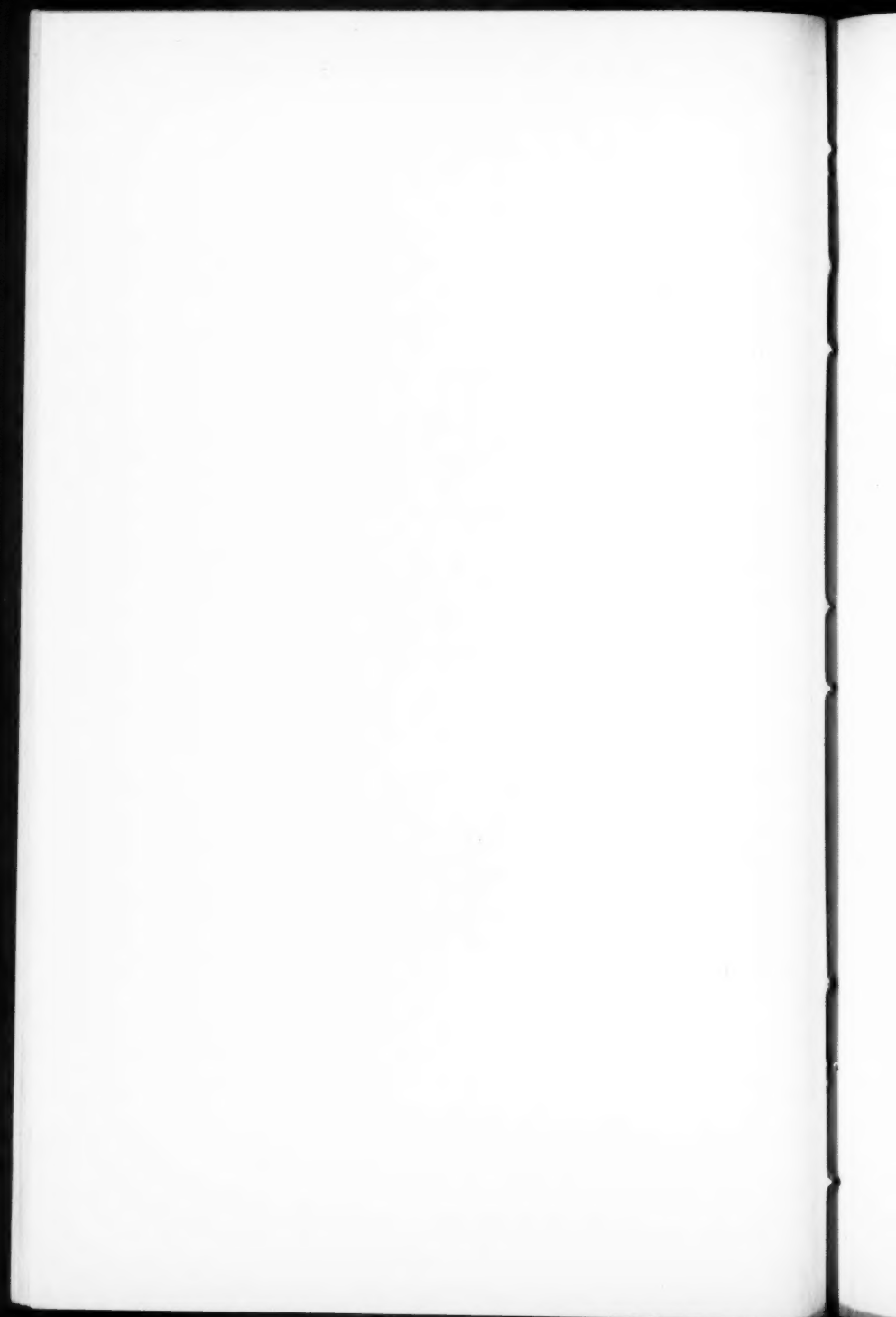
Friedländer was observed, and in about 8 of the cases a small bacillus resembling the *Bacillus influenzae*.

From the fluid material escaping from the mouth, ears, and nose of those dead of the disease cultures and spreads were made, and while a few pneumococci were observed, most of the organisms were contaminating bacteria, and some, especially in culture, were of the spore-bearing variety. None of these were organisms resembling the *Bacillus influenzae*.

A few blood-cultures, less than a dozen, were made, and these were all sterile.

From these studies it was learned that the bacillus of influenza or an organism corresponding to it in morphology was present in a very small percentage of cases, and from the commencement of the disease the infection appeared to be one of a mixed type.

I wish to extend my thanks to Mr. Louis Borow, Undergraduate Assistant, and to Dr. E. A. Case, Resident Physician, of the Philadelphia Hospital, for assistance in obtaining specimens of sputum for study and for the cultures from the throats.



## CLINIC OF DR. CHARLES W. BURR

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### THE MENTAL COMPLICATIONS AND SEQUELÆ OF INFLUENZA

A SERIOUS feature of the recent epidemic of so-called influenza was and is (for cases of the disease are continuing to occur) the large number of patients suffering from mental complications, either delirium during, or insanity after, the acute attack. There are no accurate statistics, based on large numbers, as to the frequency of delirium during the febrile stage because in no civil hospital were there assistants enough to keep any but the briefest records. The statistics of the military hospitals will, when published, be very valuable because they are based on thousands of carefully recorded cases. Conclusions drawn from a small group of cases as to the frequency of any symptoms, mental or physical, in any disease are valueless, because in one series a symptom may be too frequent, in another too infrequent. The greater the gross number of cases studied, the more nearly will the percentage of occurrence of any one symptom agree with the real percentage. We will never have accurate statistics of the percentage of insanity following the acute illness and appearing during convalescence, because many patients are treated by one physician for the influenza, and by another, who may not know the history, for the mental disorder; and this is true not only of patients treated privately but also of those in hospitals. For example, we frequently have patients brought to Blockley, picked up by the police, about whom some vague statement is made of a recent attack of influenza. We do not count them as postinfluenzal cases because we are not sure, but more or fewer of them are; others doubtless, even if recently ill, have been in-

sane for many months. We exclude, therefore, all patients who do not have a positive history, and hence our statistics show a lower percentage than some other hospitals for the insane. Notwithstanding the paucity of statistics, there is no doubt that the percentage of patients with febrile delirium or insanity has been larger in this than in any epidemic or pandemic that has appeared in America for many years. The number becoming insane has been so large, influenza has been the exciting cause of chronic incurable dementia in so many people congenitally doomed to mental breakdown, that it is possible the number of persons insane may for several years after the cessation of the epidemic be slightly reduced, because the human material available (in other words, the people congenitally predisposed to insanity and possessing cerebral cortices abnormally susceptible to the irritation of the poison of the acute infectious fevers) will have been used up. Probably no victim of this epidemic has escaped entirely all mental disorder, though, fortunately, in a very large majority the disturbance has been trifling; the patient suffers from slight mental depression, along with physical weakness during convalescence, if he escapes more serious disorders.

This is not the place to discuss causation, but probably a part, at least, of the mental sluggishness and emotional depression is caused by circulatory weakness produced by a degeneration of the heart muscle. Fever in and by itself, no matter what its cause, may bring on delirium, but, in addition to the fever, there is also in this disease a true intoxication, a local poisoning of the cells of the cerebral cortex.

The following abnormal mental conditions are found: During the acute illness, delirium or hebetude; during convalescence: 1, psychasthenia always associated with neurasthenia; 2, a state of mild elation without delusions (very rarely); 3, confusional insanity; 4, dementia præcox, alcoholic hallucinosis, and (though very rarely) paresis. Influenza is not the true cause in Group 4, but the merest excitant.

The delirium, either excited or depressed, differs in no way from that seen in lobar pneumonia, typhoid fever, or, indeed, in any acute infectious fever. It may come on suddenly, at the

very onset of the attack, before there is any involvement of the lung. This is important to remember because a superficial examination may lead to a hasty diagnosis of acute mania, whereas a careful examination and the discovery of fever would lead to a correct opinion. Physicians sometimes forget that, barring Bell's acute delirious mania, fever is not a symptom of mania, and its persistence, if at all high, means some visceral disease. More frequently delirium does not appear at the onset, but one or two days later. Its severity varies with the rise and fall of the temperature. Moderate or slight delirium does not increase the danger of a fatal outcome, nor the chance of a later serious mental breakdown. Extreme delirium is of very serious import as to life; it is usually followed by stupor, coma, and death. Severe and continuous delirium occurring in slight fever indicates that the patient is of an unstable mental make-up and probably has not a good family history. There is one curious negative exception to this rule, namely, women of the hysteric-neurasthenic type (the emaciated, intense, and highly intellectual group, who look as if they had no power to resist disease), who certainly are victims of a biologic taint, often carry quite high temperatures well, pass through a quite severe fever with minds clearer and sometimes wholesomer, and with better emotional balance, than they show when in their every-day state. Transient delirium or stupor in high fever is not an evidence of poor protoplasm, but a normal reaction.

In some patients delirium alternates with profound hebetude, or the latter may persist throughout the entire acute illness. The profundity of the mental sluggishness varies greatly. One patient may simply want to be left undisturbed, another may require great effort to arouse him enough to take his food, and may neglect the calls of nature. True coma is, of course, rare except in patients hastening toward death. There must be some physical reason why some people become mentally excited and others obtunded when suffering not only from the same disease, but when apparently they are physically in identically similar conditions, and reacting to the same morbid stimuli, some difference in the mechanism controlling chemical changes in the

body, but what it is is unknown. When we know more of biologic chemistry and when we know more of clinical psychology, when we know how correctly psychologically to classify men, we will be able to foretell that, given certain conditions, one patient, A, will be delirious, and another, B, be in stupor.

Psychasthenia and neurasthenia are usually coexistent. They are so common after influenza that it is more correct to regard them as part and parcel of the disease, a necessary consequence, rather than as complications. The course of events in an every-day case is as follows: The patient ceases to be delirious if, indeed, he has had any delirium, his temperature becomes normal, he sleeps well, begins to want food, to digest it well, and if he has had a bronchopneumonia it passes away, but instead of regaining strength, he finds himself miserably weak, physical exertion sends his pulse-rate up and causes sweating, and he neither wants to, nor can, make any mental effort. To make a decision about some trifling matter tires him, and an important matter requiring any deep thought, for even a short time, must be put to one side. He realizes his mental weakness and, as a rule, is distressed by it, though rarely one meets a genuine philosopher who does not worry. The ordinary man is either fretful and peevish, soft, or despondent. This despondency, and it sometimes occurs independently of any serious neurasthenia, any mere physical weakness, may be so severe as to be a real melancholia. I have seen two men during this epidemic who attempted suicide, seemingly because they had, without any other symptom, lost all desire to live, so profound was their depression. Certainly there was not in either patient any outside motive—no need of money, no family quarrel, none of the things that drive sane men to suicide—nor did they act under the influence of a delusion, unless it came into their minds at the moment of the attempt. In both cases no one suspected, though everyone knew they were much depressed, that they needed constant watching. As sometimes happens after attempts at suicide, one patient lost his depression almost at once; the other continued depressed for several weeks, but now seems normal. Both stated, on recovery, that they tried to kill themselves because they

were horribly down-hearted and could not convince themselves they would ever feel otherwise. Another man tried to kill himself because he had been told, what was not true, that he had not only influenza but also phthisis. He had a family to support and, brooding over his condition, decided to end it all. In his case there was an external motive, but (and I can speak, for I knew him well) he would not have dreamed of suicide had he not been made despondent by his acute illness.

The outlook as to complete recovery from postinfluenzal neurasthenia and psychasthenia is absolutely good. Even profound simple melancholia is almost always recovered from. Indeed, unless there appear delusions of self-accusation recovery is almost always certain to come after some weeks, and even with the delusions, after several months. Every patient, however, who is profoundly depressed should be watched and studied as to his true emotional state. Too often we neglect the emotional study of patients. For example, a youth twenty-two years old was admitted to a hospital with a temperature of 103° F. His fever continued between 101° and 102° F. for four days, and on the sixth day fell to normal and remained there. He had no definite signs of any extensive lung involvement, and in ten days from the onset began to sit up and walk about. In fourteen days he began to do little jobs around the ward, was quiet, talked very little to the other patients, but seemed anxious to be kept busy. (I suspect he wanted to keep occupied in order to stop thinking.) No one paid much attention to him until, on the twenty-first day after his admission, he was, by the purest accident, caught just as he was about to jump off a fire-escape at the fourth or fifth story. He was, when found, dazed and confused, and was sent to another hospital, where I saw him. On my first examination he was too apathetic for me to learn much about his mental condition, and remained so several days, needing to be spoon fed, but not unclean, not noisy. A few days later he began to talk and to blame himself because of his habit of masturbation. He constantly accused himself of sinning against God. He rapidly demented. In his case the influenza was merely the exciting cause of an adolescent insanity which was sure to

come sooner or later. I report him here as showing how important watchfulness is to prevent suicide.

Very rarely during convalescence a patient, instead of feeling neurasthenic and mentally depressed, feels a little elated and imagines he is stronger than he really is. He is not grandiose, has no delusions of grandeur, but is simply a little too happy. This abnormal emotional attitude never lasts long, and the only danger from it is that the patient may dismiss his physician too soon, overexert himself, and die suddenly from his weakened myocardial muscle being overworked, or the physician may mistake the elation for genuine good health, dismiss himself, and later be blamed for the death.

The true insanities occurring in influenza come to the surface, as a rule, during convalescence. I say "come to the surface" because they have been potentially present since conception. It is unusual for the delirium beginning in the febrile stage to continue after the fever ceases and to become chronic. Patients who are going to become insane after the acute attack, as a rule, show no very marked mental symptoms during the febrile stage; or there may be delirium, which passes off, and is succeeded by a period of mental clearness, followed by insanity. The symptoms of insanity begin to appear several days or several weeks after the beginning of convalescence. The most common clinical picture is that of confusion. There may be fleeting hallucinations of sight and hearing, periods of mental inertia interspersed with periods of active but confused thought. The patient may be able to appear, indeed be, mentally clear for a moment or two. For example, he may start to answer a question responsively, but before he has spoken many words will wander and talk incoherently. The hallucinations are usually unpleasant, indeed, horrible. Rarely they are pleasant. Patients more often hear the cursing of devils than the singing of the heavenly choir. Hallucinations of taste and smell are much less frequent than those of sight and hearing. Delusions also occur. One young married woman believed she had had a miscarriage and so accounted for being in bed. She was much confused, but always came back to the miscarriage. One moment her husband was

dead, and the next he had married a second wife, the wife being any nurse within her field of vision at the time. At times she was elated, singing and shouting; at others apathetic, emotionless. Within four weeks mental health began slowly to return. On the whole, the prognosis, both as to life and as to recovery, is good in these cases of acute confusional insanity, and, as a rule, the duration is five to eight weeks. The shorter the attack, the less likelihood of a recurrence of mental disorder, either after stress or spontaneously.

Most serious of all the mental complications of influenza are the cases in which it simply hastens, simply precipitates, the occurrence of an insanity. Youths or young adults, predestined to an adolescent insanity, a dementia præcox, are especially prone to break after an attack of influenza. I believe, though I cannot prove it statistically, that influenza, especially when it is associated with a bronchopneumonia, is much more dangerous, as an exciting cause of dementia præcox, than typhoid fever or lobar pneumonia, though even it is not a frequent cause, most cases of dementia præcox arising from endogenous, not exogenous, causes. Usually whatever type of adolescent insanity appears as the final state, the beginning is confusion; later there may be catatonia or a paranoid dementia. All of the patients presenting a true insanity whose histories I have been able to get showed a family taint; either insanity, hysteria, epilepsy, or degeneracy of some kind had been present in one or more of their near kin.

Influenza seemed to be the exciting cause in two cases of paresis or, rather, seemed to bring out the mental symptoms. Both patients were young men, one thirty-eight the other thirty-five years old; one had had a chancre five years before; the other, three. The families, in each case, denied the existence of any sign of mental disease before the acute illness appeared. Both the patients had grandiose delusions and were demented. One had scanning speech and increased knee-jerks, and the other had pupillary inequality, with poor reaction to light and exaggerated knee-jerks. Both had the paretic facies. The physical signs, caused by spinal cord degeneration, could not have been the result of the influenza, and must have existed before the onset.

I am forced to believe that both were cases of paresis beginning with physical symptoms and that the influenza hastened the appearance of the mental symptoms.

I have seen quite a few children from eight to twelve years old, all of whom were delirious, and five of whom died with pneumonia during the delirium, or, rather, in a consequent coma. In several of the children there were distinct signs of meningitis, and had I seen them in the epidemic of poliomyelitis in 1916, I should have diagnosed their cases as being instances of the meningeal type of that disease. I may add, though it is a little away from the subject of this paper, I have seen several children who, without mental symptoms, showed the spinal-cord-symptom picture of the same disease. Have there really been two or three different epidemics running concurrently, or does influenza do all the remarkable things seen this year? The first assumption is the safer. I can draw no conclusions as to the frequency of mental involvement in children suffering from influenza, because I have only an opportunity to see those mentally or nervously affected.

Let me say a few words as to the effect of influenza on people already mentally defective. I have seen four imbecile children who were attacked. One, a girl fourteen years old who had been simply feeble-minded, became violently delirious during the fever, hallucinatory and delusional later, and finally had to be sent to an institution, where she now (three months later) remains. She is dirty, destructive, foul-mouthed—a complete imbecile. The other three were children from eight to ten years old, who, though feeble-minded, had not needed institutional care. They were mildly delirious at first, and so much degraded later that they could not be kept at home. In all, therefore, the influenza caused a very rapid and permanent increase in the already present mental weakness. This is not always the case; many imbeciles, I have been told by physicians in institutions, pass through an attack and are the same after as before.

It is an interesting fact that patients suffering from what we call chronic mania, as a rule, or at least it has been my experience, become stupid and apathetic during the fever. Off hand, one

would expect they, of all men, would become delirious. On the contrary, the fever, or the poison of the disease, has a sedative instead of a stimulating or irritating effect on them. Most of those whom I saw, who survived, are in almost the same mental state they were in before; a few are distinctly demented. Paretics, who have mind enough to be influenced, seem to be affected in the same way as maniacs. It is important to keep a keen eye on the insane at all times, and especially during an epidemic, lest they be neglected while ill, because they are prone to a mental anesthesia of the viscera, in consequence of which they may be attacked by inflammatory or other pathologic processes which would cause pain in the normal man, without showing any sign of suffering, without any complaining.

Alcoholics (and let me say by that word I mean the congenital degenerates who have an abnormal craving to drink to excess, and the silly fools who, being lazy, get drunk constantly because they have nothing better to do, not sane men, who use liquor sanely) do not withstand any acute infection, including influenza, well. They are prone to develop delirium tremens or some other type of alcoholic psychosis, and the outlook as to life is always bad.

The treatment of febrile delirium resolves itself into a question as to whether, in a given case, the delirium or the sedative will do the more harm. Measures to reduce the fever, if high, are, of course, always indicated. Frequent sponge-baths are very useful for this. The same antipyretics are used as in any febrile state. Sedatives are contraindicated unless the delirium is so intense as to be really exhausting the patient and preventing sleep. The old-fashioned opium mixtures are still useful in this condition. The synthetic drugs, especially those which depress the heart action, should be used only when absolutely needed.

The sheet-anchors in the treatment of postinfluenzal psychoses are food and rest, followed by food and exercise. It is doubtful if the old explanation, of mere exhaustion alone, explains the mental disorders following the acute infectious fevers, but rest and quiet are most important elements in treatment. In conditions of mental excitement the continuous warm bath

often has a very quieting effect. The patient pays no attention to his bowels, and hence constipation is common. This must be overcome by some laxative that will not purge, but cause one or two stools, and the more nearly normal the stools are in color, consistency, and amount, the better. Continued purgation is harmful. When the patient will co-operate in the matter of eating, he may have a mixed diet, with milk between meals and at night. It is necessary to remember that many patients will not chew food, and such cases must be kept on a mushy and liquid diet. When the patient is in a condition of simple stupor, or catatonic, he must be fed by the nasal tube. One quart of milk, two ounces of sugar, and two eggs, beaten up together, and given twice daily, will give sufficient nourishment. During the period of physical weakness massage is very useful. So soon as the patient is physically able he should be given exercise out of doors. If he can be induced to enjoy any outdoor sports he is on the high road to recovery. It is useless, indeed harmful, to attempt mental stimulation by mental exercise until the patient is well enough to co-operate.

CONTRIBUTION BY LIEUT. EUGENE A. CASE, M. C.,  
U. S. N. R. F.

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**BACTERIOLOGY OF INFLUENZA**

THE pandemic of influenza just passed is but a repetition of similar ones, though perhaps more virulent than most of them, and it will be interesting to pause for a moment to glance at the explanations given for the earlier visitations of the scourge.

In the absence of exact knowledge as to the cause of disease it is but natural that those who practised and taught the art of medicine should be influenced by various natural phenomena occurring coincidently with epidemics, and should have given rather free rein to theorizing, a characteristic by no means rare at present.

In 1510 and even as late as 1762 the appearance of a comet at the time of an epidemic was looked upon as more than a mere coincidence.

Volcanic eruptions, such as those of *Ætna* in 1556, 1675, and 1762, *Vesuvius* in 1737, *Hecla* in 1781, with their discharge of noxious vapors and dust; offensive fogs, as in 1537, 1775, 1782, and 1831; damp cold winters following unusually hot and dry summers; swarms of flies and locusts; distempers in horses and cattle—all these were considered in the etiology of influenza, and careful studies have been made of the meteorology of various sections with the purpose of determining whether or not the weather was an important factor.

It was pointed out by some, however, that the rapid and universal spread of the disease and its existence in various climates, favorable or otherwise, rather militated against this theory. The present epidemic has shown very conclusively that climate had no part in either its production or its spread.

In 1850 John Groves,<sup>1</sup> of London, published as his opinion that a living germ was the cause of the disease.

Peacock<sup>3</sup> thought contagion played an occasional part in the spread of influenza, but by no means the most important one.

One writer, Basle, thought ozone a considerable factor in the production of the disease.

With the demonstration of micro-organisms as the cause of infectious diseases it became clear that epidemics depended upon the rapid spread of these organisms from individual to individual, and in the case of influenza they spread along the lines of human travel and at about the same rate.

At the time of the epidemic of 1889-92 many men whose names are now familiar to every physician and who founded the science of bacteriology, worked upon the problem, but were unsuccessful until Pfeiffer demonstrated the bacillus which bears his name in 1892.

It is interesting to note the resemblance in the reports of the studies of that epidemic and the present one.

Dr. Kline<sup>3</sup> examined a number of blood-films, made 43 blood-cultures, and believed that he found a minute bacillus in all of his cover-slip preparations and in six of his blood-cultures. Others making similar experiments were unable to confirm his results, and during the present epidemic blood-cultures have been uniformly negative so far as the *Bacillus influenzae* is concerned.

Kline also cultured the bronchial secretions, using a slightly alkaline broth, and was able to confirm Pfeiffer's discovery, finding the bacilli most numerous in the early stage of the disease. His experiments with animals were negative. In one autopsy he isolated the influenza bacillus.

P. Canon<sup>4</sup> felt convinced that he had demonstrated the bacillus in cover-slip preparations, and was supported by Koch, but J. H. Wright,<sup>5</sup> of Boston, pointed out the very obvious possibilities for error in his work. Pfeiffer and others were never able to demonstrate the bacillus in blood-films.

Klebs,<sup>6</sup> in studying the blood from cases of influenza, found "an enormous mass of small, actively moving, highly refractile corpuscles," and thought because of the recurrences or relapses in the disease that it might be due to a protozoan.

Ribbert,<sup>7</sup> studying the lungs from cases of influenza, found the streptococcus, especially in lungs where no consolidation was present.

Weichselbaum<sup>8</sup> found the pneumococcus in the sputum in nearly all cases, in the urine in one case, in inflammations of the accessory nasal sinuses, in otitis media, and in one case of a complicating meningitis. He was unable to isolate anything from the blood. He believed the cause to be an organism as yet unknown, but felt that the pneumococcus played an important rôle as a secondary invader.

Babes<sup>9</sup> described two organisms which he called Bacterium No. 1 and Bacterium No. 2. The former, a minute bacillus, was Gram-negative, formed fine rods, or was even pear shaped.

His Bacterium No. 2 was somewhat similar in morphology and growth, though larger, and Gram-positive.

Finkler<sup>10</sup> describes pneumonias similar to those seen in this last epidemic, speaking of the consolidation as being splenized rather than hepatized. He secured the streptococcus from the lungs, and in only one case a staphylococcus and a diplococcus, not the pneumococcus.

Levy,<sup>11</sup> studying the cases at the Strassbourg Clinic, found in the sputum, in pus from otitis media, and from the pneumonias pneumococci, streptococci, and staphylococci, but did not believe them to be the cause of the disease.

R. Pfeiffer<sup>12</sup> in January, 1892, summed up his discoveries as follows:

1. A definite species of bacilli found in all cases of influenza studied. In uncomplicated cases they were in pure culture and in immense quantities as a rule. They may penetrate the bronchial tubes and invade the peribronchial tissue, and even reach the pleura, as in one case.

2. They were found exclusively in influenza cases.

3. Their presence kept pace with the course of the disease, decreasing in number as the patient improved.

4. He had seen and photographed similar bacilli in enormous quantities two years ago.

5. They were small rod-like organisms that sometimes grew

in short chains, and did not stain well with most of the aniline dyes. They were immobile, and because of their peculiar staining some of the earlier observers who evidently saw them took them for diplococci.

6. They grew on 1.5 per cent. sugar-agar as extremely small, clear droplets that could not be seen at times without a lens. He could not cultivate them beyond the second generation.

7. Inoculation experiments were conducted with apes, rabbits, guinea-pigs, rats, pigeons, and mice, but were positive only in rabbits and apes.



Fig. 139.—*Bacillus of influenza*. Smear from sputum (after Heim).

8. He felt justified in calling this organism the cause of influenza.

9. The infection is spread by way of the sputum.

Kitasato<sup>13</sup> thought it remarkable that the organism had not been discovered before, but believed this to be due to the extreme difficulty in securing a pure culture.

The *Bacillus influenzae* is small ( $0.2 \times 0.5 \mu$ ), possesses no flagella, is not motile, and forms no spores (Fig. 139).

It is best stained with a 10 per cent. aqueous solution of carbol-fuchsin or with hot Löffler's methylene-blue. We found a useful routine method to be Gram's, followed by 10 per cent.

carbolfuchsin as the counterstain. This brings out both the organisms positive to Gram's and those negative, the influenza bacillus being among the latter.

They are commonly very numerous in the sputum from an early case, and appear as minute rods suggesting diplococci at times because of the polar staining. In preparations made from cultures they show variations in size and shape, being much less uniform than in the secretions from the respiratory tract.

The earlier workers used glycerin-agar and nutrient broth for cultivating them, but had great difficulty in keeping the organisms alive for more than two generations. Pfeiffer later discovered that they grew best when blood was added to the media. They belong, therefore, to that group of organisms known as hemoglobinophilic, *i. e.*, organisms that require hemoglobin for their best development.

A satisfactory medium is nutrient agar titrated to plus 0.5 per cent., to which 0.5 per cent. of defibrinated, unlaked human or rabbit's blood is added.

Another is nutrient broth with the same reaction, containing 1 per cent. of sodium citrate and a small amount of human or rabbit's blood.

We have found these exceedingly useful media not only for the influenza bacillus but also for the streptococcus, pneumococcus, and (when sugar is added) for the meningococcus also. Sheep's blood may be used and it may be laked, though we have thought the unlaked blood to be more satisfactory.

In isolating the organism from sputum or other secretions the material is first planted on blood-agar plates, incubated for twenty-four or forty-eight hours, the plates carefully examined, and the favorable looking colonies picked.

These are planted in broth containing a small amount of blood and 1 per cent. sodium citrate.

After twenty-four to forty-eight hours the broth culture is smeared over a blood-agar plate with a sterile swab and incubated. If the influenza bacillus was present in the colony picked, it can be easily isolated in pure culture by this method.

On agar the colonies are minute, colorless, and may escape

detection unless the plate is very carefully examined, using a lens.

With a heavy growth the colonies can be seen quite readily, especially if the plate is held at the right angle to the light. The colonies remain discrete (Fig. 140).

In bouillon containing blood the bacteria commonly form small "woolly" masses usually lying on the surface of the blood, though they are also found at the surface of the media. When



Fig. 140.—*Bacillus of influenza*. Colonies on blood agar-agar. Low magnifying power (Pfeiffer).

the culture is young the media is perfectly clear, but later clouds slightly.

The bacteria grow best at  $37^{\circ}$  C., and are very susceptible to unfavorable conditions, such as extremes of temperature and drying.

Pfeiffer recovered the organisms from cases of true influenza, grew them upon artificial culture-media, transmitted the disease to monkeys by inoculating their nasal mucous membranes with cultures of the bacillus, and was able to recover the organism

from the sick animals. The organism has, therefore, been accepted as the cause of influenza.

Because of the frequency of relapses and subsequent reinfections it has been believed that immunity was not conferred by an attack. Experiences during this past epidemic, however, raise the question of immunity afresh, as most of the individuals attacked were under forty years of age.

This may be explained by an immunity developed as the result of a previous attack, though the question cannot be satisfactorily answered as yet.

The disease is extremely infectious, the contagion being spread through the secretions of the respiratory tract, discharged by coughing, sneezing, or spitting.

Because of the rapidity of the spread of the disease, the short incubation period, and the wide-spread infection quarantine measures proved to be impracticable. In one western city the wearing of masks was compulsory, and it will be interesting to learn if this practice reduced the number of cases.

The use of masks by those in contact with patients is to be recommended, though the masks must be properly made and other precautions taken, such as care in adjusting the mask, cleanliness of the hands, etc.

In the earlier part of this paper a few reports from an extensive literature on this subject were quoted, and show the failure of many bacteriologists to demonstrate the influenza bacillus. Other organisms were isolated, such as the pneumococcus and streptococcus, especially in fatal cases and in the pneumonias, a fact of striking interest in view of the findings during the present epidemic.

Little, Garafallo, and Williams<sup>14</sup> failed to find the *Bacillus influenzae*, but did find a Gram-negative diplococcus in all of their cases.

Gotch and Whittingham<sup>15</sup> found the *Bacillus influenzae* in 8 per cent. by cultural methods and in 62 per cent. in stained smears from the sputum, and attributed the infection to a Gram-negative coccus "perhaps in association with the influenza bacillus."

Averill, Young, and Griffith<sup>16</sup> were able to cultivate the Pfeiffer bacillus from the sputum.

Matthews,<sup>17</sup> using a trypsinized blood-agar medium, found this organism in all of 12 cases studied. His cultures were taken from the nasopharynx by means of the West tube.

Martin<sup>18</sup> found the influenza bacillus in smears and cultures, and believes the epidemic to be true influenza.

Krumbhaar,<sup>19</sup> in a short note to the *Lancet*, takes issue with Little, Garafallo, and Williams, finding the influenza bacillus in three-fourths of a small group of cases.

Keegan,<sup>20</sup> in this country, had rather unsatisfactory results from sputum cultures and mouse inoculations, but found the influenza bacillus in some of his cases. In cultures from the lungs, however, in 23 cases, taken at autopsy, he was able to isolate the organism in 82.6 per cent., 31.6 per cent. being pure cultures.

Nuzum, Pilot, Stangle, and Bonar<sup>21</sup> found the influenza bacillus in only 8.7 per cent. of over 2000 cases, a study conducted at Chicago. The most important and the predominating organism was the pneumococcus, though the streptococcus, staphylococcus, and *Micrococcus catarrhalis* were present in varying numbers.

Synnott and Clark,<sup>22</sup> at Camp Dix, found the influenza bacillus in the majority of cases where searched for in association with the *Micrococcus catarrhalis*, *Streptococcus hemolyticus* and *viridans*, and the various types of pneumococci. They did not find the influenza bacillus in the blood, which agrees with the experience of others.

At the United States Naval Hospital, League Island,<sup>23</sup> the influenza bacillus was isolated in pure culture in but 4 per cent. of cases, though it was present in a larger percentage in other plates. Blood-cultures were negative so far as the influenza bacillus was concerned. In one case a Type 4 pneumococcus was isolated from the blood. Various types of pneumococci, streptococci, and micrococci were found in the cultures examined.

The pathology of influenza is concerned not only with the effects of the influenza bacillus and its toxin, but with the

damage resulting from secondary invasion of other organisms as well.

The latter, in fact, account for the large majority of the serious consequences.

The toxin of influenza acts somewhat in the nature of an aggressin, breaking down the body defenses and rendering secondary invasion much easier and more serious. For example, Type 4 pneumonias have been more virulent when associated with influenza than when the primary and only infection.

The mucous membranes are intensely congested, especially the conjunctivæ and those of the respiratory tract. Epistaxis is common, and in rare cases bleeding from the stomach and bowels has been reported.

Coryza with mucous discharge is not a common symptom, though frequent in former epidemics.

The inflammation of the lower respiratory tract soon changes from the congestive stage to that of cellular infiltration and excessive mucous formation, and the cough, at first dry, is now accompanied by a mucopurulent discharge.

This inflammatory change involves the bronchi as well as the larynx and trachea, and in severe cases of bronchitis bronchiectasis may occur.

From inflammation of this sort it is but a step to invasion of the lung, with the development of consolidations. The pneumonia which occurs is usually lobular and bilateral, though much more marked in one lung than in the other. The right lower lobe is most frequently involved, but in fatal cases this is but the forerunner of involvement of the other lobes.

The lesion begins in one or more foci and extends until practically an entire lobe is affected, sometimes an entire lung. The pleura shows well-marked pleuritis, which in some cases is characterized by an extensive fibrinous or serofibrinous exudate. The purulent form (empyema) occurs as a sequel rather than as a coexistent complication.

In this epidemic it has not been as common as during the pneumonia epidemic of last winter.

The appearance of the cut surface of the lung varies with the

character of the infecting organism and the age of the various processes. In one case the lung presented a mosaic of dark red, moist, air-containing areas, dark red glazed consolidations, reddish-gray granular ones, others similar to gray hepatization, and still others showing very early resolution. In this case the lesion was, anatomically, lobar in character, but it was essentially a lobular pneumonia that by extension had involved the entire lobe.

Pericarditis and endocarditis have been reported, though lesions of the cardiovascular system of this sort are not common. The toxin has undoubtedly a pronounced effect on the cardiac mechanism, as can be observed in the relatively slow pulse, the low blood-pressure, and the cyanosis which is so common in the severe cases.

Evidence of severe irritation of the kidneys is seen in the large number of cases showing albumin and casts in the urine. The kidneys examined at autopsy show acute parenchymatous nephritis.

Severe lesions of the gastro-intestinal tract are uncommon, but definite cases of enteritis with hemorrhage have been reported.

The liver shows cloudy swelling and the spleen is frequently enlarged, sometimes being of the so-called "splenic tumor" type.

Middle-ear disease with its attendant dangers and complications has been prominent in earlier epidemics, and is not rare in this one. The same is true of lesions of the accessory nasal sinuses. In these conditions a complicating organism is usually recovered.

Myositis, arthritis, encephalitis, myelitis, neuritis, and psychic disturbances, such as melancholia and mania, have been described.

Considerable doubt has been expressed as to the etiologic relationship of the influenza bacillus with the epidemic just passed, founded upon the contradictory reports given by various workers. While the investigation as a whole has been very unsatisfactory, nevertheless, taken with the clinical history, we feel justified in believing this true influenza.

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CONTRIBUTION BY DR. J. LESLIE DAVIS

**NOSE, THROAT, AND EAR AFFECTIONS COMPLICATING  
OR FOLLOWING THE RECENT EPIDEMIC OF SO-  
CALLED INFLUENZA, WITH A VENTURED INTERPRE-  
TATION OF THEIR SIGNIFICANCE**

FROM an incomplete review of references to nose, throat, or ear phenomena in recent discussions on the influenza epidemic, whether in official or individual reports from large army, naval, or civilian hospitals, or whether from observations by private practitioners in large cities or isolated rural districts, regardless of longitude, latitude, or altitude, there seems to be a common agreement that practically all cases of so-called influenza, at some stage, if not throughout the course of the disease, manifested symptoms, varying from slight discomfort to marked distress, of the upper respiratory tract.

The most characteristic feature of these conditions, however, has been their utter lack of any characteristic order, mode of invasion, or degree of persistence. There was no group of symptoms nor even one symptom manifested in all cases, though collectively those commonly observed were nasal congestion, epistaxis, headache, sinusitis, pharyngitis, laryngitis, otitis media, and mastoiditis. In other words, there evidently exists no upper respiratory syndrome peculiar to the disease or in any wise different from those manifested in ordinary infections or irritations of the same structures. Two of the most common symptoms occurring in the early stages, one objective and the other subjective, and of particular interest in this connection, are headache and epistaxis, each of which may be accounted for as a purely mechanical process and probably very largely dependent upon another frequently associated condition, namely, low blood-pressure.

Observations for some years have led me to believe, regardless of the exciting or contributory influences, that the direct mechanical cause of the majority of headaches, at least in their early stage, is located in the upper or ethmoidal area of one or both nasal chambers, being either a pressure irritation of the nerves, or else a toxic irritation through interference with sinus ventilation. This may be due to the constant existence, in those habitually predisposed, of some form of obstruction (deflected nasal septum, enlarged or misplaced middle turbinates, granulations, or polypoid growths, etc.), further aggravated in each attack by toxic absorption from diseased tonsils, acute or chronic indigestion, intestinal auto-intoxication, or marked atmospheric changes, etc.; or else, in those not so predisposed, it may be due to the intensity of some acute process causing marked congestion or swelling of the membrane in the ethmoidal region.

I have seen numerous cases of headache relieved temporarily by reducing the swollen, intumescent or engorged nasal membrane with applications of an astringent solution, and its permanent relief when the local reduction of pressure was followed by correction of the predisposing constitutional or systemic factors without resort to any local surgical procedure. Of these, intestinal toxemia is perhaps the most common, resulting frequently from a condition of hypocholia, which, in turn, is likely the result of toxic absorption from some focal infection of which the faucial tonsils are the most common and most constant offenders.

The premonitory headache in certain acute infectious diseases I believe to be mechanically produced in the same manner, its severity depending upon the degree of existing local predisposing factors; its persistence depending upon the prolongation of the toxic source or else the possibility of an ensuing sinus infection.

The phenomenon of epistaxis, likewise, may have both a local and a constitutional predisposing element. For instance, probably 90 per cent. of such hemorrhages emanate from a small area on either side of the nasal septum near the anterior margin, where the mucous membrane is prone to become dry and, in the case of more or less catarrhal irritation, "crusty," so that small

branches of the septal artery which run exceedingly near the surface at this point may be easily ruptured by cracking of the membrane in this dry space. A somewhat similar process may take place in the early, dry, congested stage of an acute rhinitis, and, once started, may persist or recur at intervals according to the continuation of certain constitutional factors, such as pyrexia, toxemia, blood-pressure, etc.

Thus my object in dwelling upon these symptoms, headache and epistaxis, commonly referred to in influenza reports from all sections of the country, is to emphasize the mechanical processes engaged as found commonly recurring in the initial stages of various ordinary acute infections. And even all the other symptoms enumerated seem to be in nowise different from the same phenomena occurring in the beginning or during the course of the common affections or infections of the upper respiratory tract and its closely associated structures.

For the purpose of comparing and contrasting certain phenomena of the epidemic and those of a similar character observed at other times, I wish to quote from several recent reports from large Army, Navy, and Civilian Hospitals.

These extracts had to be taken more or less at random from general reports, since up to the present time I have been unable to find any published observations especially on the subject of nose, throat, and ear complications or sequelæ.

Alfred Friedlander et alia, in the Camp Sherman report (*Jour. Amer. Assoc.*, November 16th), state: "Other than pneumonia, complications to influenza were slight. Profuse epistaxis without nasal ulceration was very frequent at onset, and later acute catarrhal otitis media was common, but fortunately always cleared without perforation of the drum. Hemorrhages into the middle ear and a few instances of acute sinusitis occurred."

From Lieut.-Commander Daland's recent discussion before the College of Physicians, on characteristics observed at the Philadelphia Naval Hospital, is the following: "Onset variable, may be slight coryza, dry cough, loss of voice, epistaxis, pains in head, back, and limbs, slight fever, marked circulatory de-

pression, intense toxemia affecting vasomotor and cardiac apparatus."

Bernard Fantus, Rush Medical College, Chicago: "One of the startling features of the pandemic was its sudden flaring up and its equally sudden decline. Among those who escaped well-marked sickness there are few who could not recall having had an occluded or running nose, a raw feeling in the throat, or a cough or ache or pains at some time during the period of prevalence of the disease. In view of the universal prevalence quarantine seemed useless and face masks were not prophylactic—nurses who wore masks were largely susceptible. Nose not as frequently affected as bronchial tubes; when it was involved, showed marked tendency to bleed. Throat rarely complained of."

Nuzum and Associates, from the Laboratory of Pathology of the Cook County Hospital, and the Department of Pathology in the University of Illinois College of Medicine. A bacteriologic study of the prevailing epidemic especially among the civilian population forms basis of paper: "Among complications of the disease, 11 patients developed a unilateral or bilateral purulent otitis media. Pure cultures of pneumococci were isolated from 8 discharging ears, the hemolytic streptococcus in 2 patients, and the *Streptococcus viridans* in the remaining case. One child developed an acute and fatal mastoiditis. Purulent frontal sinusitis was encountered in one instance at necropsy, and cultures yielded pure hemolytic streptococci. It is to be expected that sequelæ of still more diverse nature may subsequently develop."

Strouse and Bloch, Michael Reese Hospital, Chicago: "Among initial symptoms are conjunctivitis, more or less severe coryza, headache, chilly sensations, and elevated temperature; a normal or slightly diminished systolic blood-pressure in those patients in whom the disease ran a moderate course; the diastolic pressure, however, was considerably lower than was to be expected. A systolic pressure of 100 with a diastolic pressure of 40 was not an uncommon occurrence. One patient whose systolic pressure was 120, diastolic 85, with Cheyne-Stokes breathing during the

pneumonic phase, had a systolic pressure of 180 and a diastolic of 115 on recovering from the pneumonia."

A. Francis Coutant, Chief Surgeon Hospital Ship, Manila: "During the month of June, 1918, an epidemic of influenza appeared in the Philippine Islands, which presented an interesting number of manifestations. About 300 cases, the majority of which were hospital patients, came directly under observation. The epidemic, so far as can be determined, began in Manila, although the fact that cases were first noted among longshoremen and other laborers along the water front might indicate that it has been brought in from some other part of the world. Within three days after the first cases were noticed between 70 and 80 per cent. of the longshoremen, clerks, and other workers at the docks were unable to work. In one large business firm 80 per cent. of the native employees were away from work for at least two days, while 50 per cent. of the Europeans were absent for one day. Within ten days after the initial outbreak the majority of those who had been ill were well enough to be back at work, and business and industrial activities resumed their normal course. However, the hospital continued to be overcrowded with cases diagnosed as influenza for two or three weeks more, and it was not until the advent of a typhoon, which brought with it a great deal of wind and rain, that the last vestiges of the disease were swept away. Frequent nausea and vomiting, and hemorrhage from mucous membranes were noted."

The following unpublished notes supplied by Major Hamburger, who was Chief of the Medical Service at Camp Zachary Taylor during the epidemic; and by Captain H. E. Happel, who was at Camp Sevier (though each is now at Camp Crane), are of special interest, reporting greater severity in nose, throat, and ear complications than from any published report I have seen.

Major Hamburger states that in the early stages of the epidemic there was almost always epistaxis, often profuse, and in the cases coming to autopsy in this stage there was practically always marked congestion of the sinuses. This was so constant that he regarded it as a part of the disease rather than a compli-

cation. Later in the disease, when the body reacts to the infection, this becomes a purulent sinusitis, not detected by the usual clinical symptoms, but found by close examination, suggested by the fact that cases coming to autopsy in this stage always showed purulent involvement of all the sinuses. He saw no cases of brain abscess or sinus thrombosis or purulent meningitis, although meningismus was noted in some cases. Otitis media was a late complication after the patient was convalescing from his bronchopneumonia. In the early days of the epidemic, he states, patients died in two or three days, consequently pus did not have time to form, but later in the epidemic the virulence became lessened and cases ran the entire course, with purulent otitis media and mastoid complications.

Captain Happel's report of conditions at Camp Sevier emphasized the frequency or almost constant purulent pan-sinusitis that was found postmortem in the early stages of the epidemic, and later observed in the living patients. He suggested that its earlier oversight might have been due to the fact that their chief attention was engrossed by the quickly developing and often fatal bronchopneumonia. He was transferred to another camp about the time the epidemic subsided, but up to that time very little otitis media had occurred, though recurrent suppuration from old lesions were noted. He emphasized the rarity of intracranial infections, and mentioned one patient who developed a left-sided hemiplegia and was trephined with the expectation of finding an abscess, but proved to be perfectly clean. At post-mortem it was found that hemorrhage had occurred in the internal capsule, probably from septic embolus. There was pus in every sinus, in the mediastinum, and in both pleural cavities. Severe headache was a common early symptom.

Thus it seems evident to me, both from statistical reports and from our personal observations, for which every physician surely has had ample opportunity, that some interpretation other than the ordinary theory of contagion must be sought in order to explain the development, the universality, and the varying degrees of virulence of the recent and most terrible pandemic of modern, if not of all, time.

In the first place the onset of the disease was more variable than is usual in any known specific infection. Varying symptoms prevailed in different localities and in different individuals of any one locality. No specific organism has been detected common to all, but, instead, the usual group of bacteria common to infections of the respiratory tract, particularly the streptococcus, pneumococcus, staphylococcus, and the Pfeiffer bacillus of influenza, were found in varying orders of predominance. The most frequent and most serious complication was bronchopneumonia, which developed quickly and terminated quickly in the fatal cases, while those that lingered in most cases recovered, or else died of other complications. Sinus and aural complications were perhaps not more frequent, except in certain localities, than during occasional previous periods of markedly unfavorable atmospheric states when "colds," tonsillitis, laryngitis, or bronchitis prevailed to the point of being spoken of as "epidemic," and when present differed apparently but little, even in degree, from ordinary sinusitis and otitis media. Regarding these conditions as sequelæ it is, of course, too early to speak authoritatively, but it seems at the present time that the percentage is far below that of any previous epidemic of influenza. It is certainly true with respect to mastoiditis.

One important characteristic of the epidemic that was common to each locality was the precipitateness of invasion of the disease following a short interval, from one to several days, after the first cases made their appearance, seemingly a day upon which every influence favoring the development of the disease seemed to work in conjunction, so that it was not an unusual occurrence in large business establishments in some localities to find 50 per cent. or even more of the employees taken down, regardless of whether their occupations had kept them out-of-doors or indoors, and seemingly regardless of whether they had come in contact with anyone already infected. This was also just as true in sparsely populated country districts, where perhaps a whole family, entirely isolated, would be similarly seized. Many of those so affected, however, recovered in the course of three or four days sufficiently to return to work; and many died

within the same short space of time, while others developed various complications that ran a prolonged course, some of which proved fatal, though a higher percentage of the protracted cases recovered.

A fact which must not be overlooked is that during the period of the epidemic there were also numerous individuals who did not contract the so-called influenza, who suffered practically the same group of nose, throat, or ear discomforts lasting from a few hours to days or longer.

From my own observations, entirely in private practice, I have been unable to see that these phenomena of the upper respiratory tract, whether occurring in an individual whose illness was diagnosed as influenza or otherwise, differed materially from those that I have commonly observed under somewhat similar atmospheric conditions, except in point of degree.

Thus, are we to believe that the rhino-laryngo-otologic characteristics, observed in a measure universally, are a part of the disease, a predisposing or etiologic factor, a complication, or merely a coincidence? For my own part I believe that they are a part of the "disease," just as much a part as are any other phases connected with it. In other words, I am about persuaded that at the present time the most rational basis on which to explain the development and spread of the epidemic is that of a world-wide meteorologic disturbance, through the obscure influences of which all ordinary bacteria commonly present in the usual infections of the upper respiratory tract were rendered more highly virulent, while through the same influences normal human resistance to these organisms was diminished. Or, instead of these two influences working simultaneously, the meteorologic conditions might first work their effect upon the human tissues, so changing the chemistry of their secretion that there is developed a culture-media in which the bacteria, already present, are speedily propagated beyond the power of resistance, and the varying degrees of disintegration of the tissues ensue.

A seemingly trivial incident might be related in this connection illustrative of the physical effects of certain atmospheric

states on organic substances. Doubtless all, at least those who have lived in the country, are familiar with the commonly observed phenomenon of milk "turning" suddenly "sour" during the course of a summer thunder-shower, the popular supposition being that it is the thunder that works the change, when in reality electric phenomena that produce the lightning and the thunder in the atmospheric media is but the common force that effects the chemical change in the milk.

Now an interesting example of the principle from a recent personal experience might be timely in this connection, since I should like to obtain further evidence of its existence elsewhere. During the height of the epidemic my attention at home was called to the fact that the milk and cream seemed not up to the usual standard of freshness, and when no improvement resulted after several complaints (notwithstanding that the dairy from which it was delivered was undoubtedly one of the best in the city), our order was transferred to another dairy, with the result that their products showed the same defect as the first; then after a few days' trial another change was made, with what seemed at first but slight improvement, though followed by a gradual change for the better, till finally about two weeks from the time the first trouble occurred the milk and cream became again consistently good. It did not occur to me till three or four weeks later, however, on finding that many others had the same experience, that the incident might in any wise have been associated with the same disturbance of atmospheric elements which at the same time possibly was favoring the devastations then being wrought on human tissues through the medium of bacteriologic organisms and products rendered more highly virulent.

It should be noted that one of the most common and most active symptoms associated with the disease in this epidemic was toxemia.

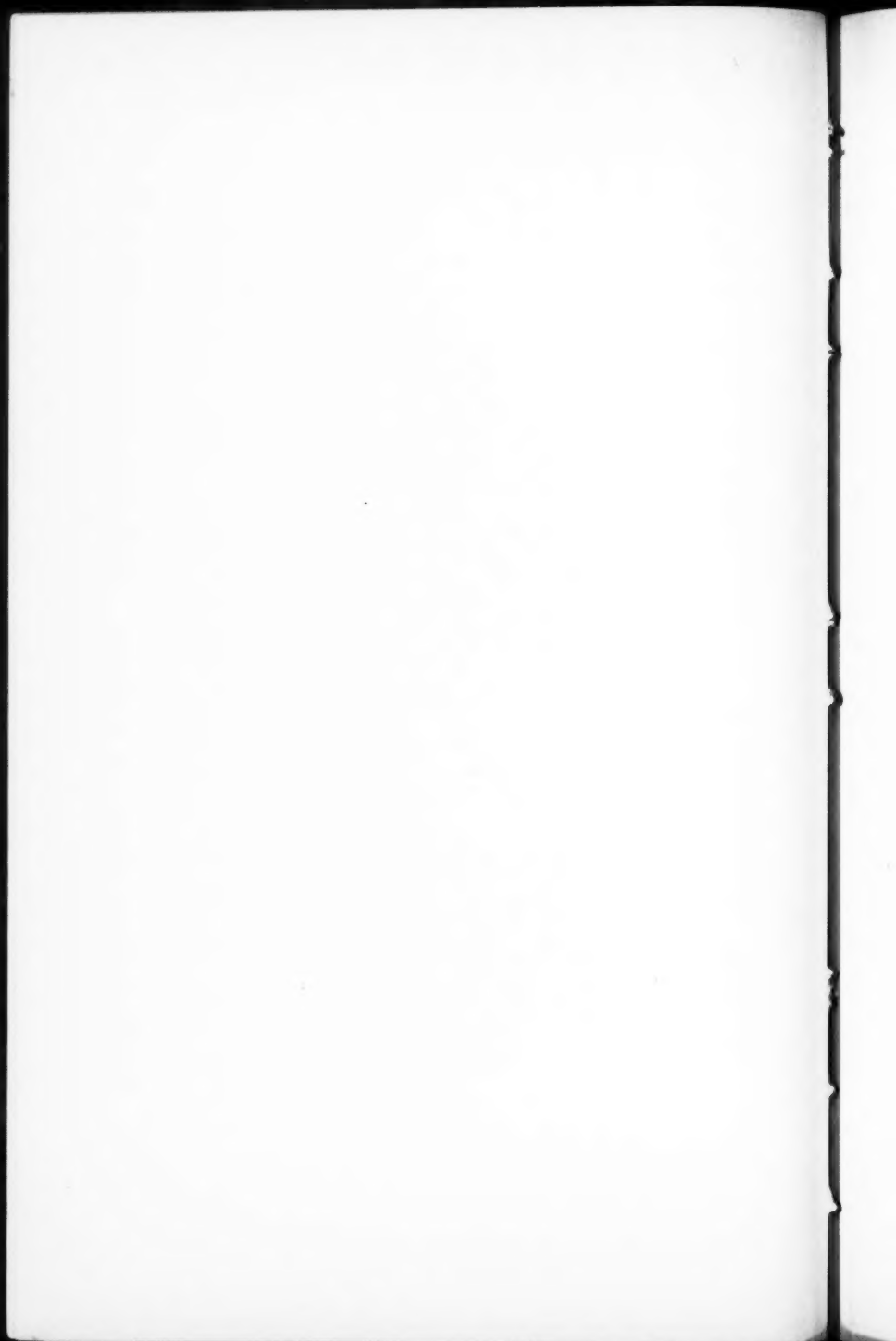
I have emphasized on several occasions during the past year the toxic influence of diseased faucial tonsils, its marked inhibitory effect upon the hepatic function, with the resulting auto-intoxication and lowered blood-pressure, which, in turn, completes the circle by producing a low-grade congestion or intumescence of the

lining membrane of all the upper air passages and accessory cavities, thus perpetuating the most favorable environment in which bacteria, that same group which have been uniformly associated in the so-called influenza epidemic, can freely generate their varied cultural characteristics. I take it, then, as a self-evident conclusion, regardless of what may be the prime factor favoring the highest degree of susceptibility of any individual to the "influenzal" infection, that the existence of badly diseased tonsils implies at least an important accessory influence.

Having observed with great interest for many years, though for the most part unintelligibly, the marked influence of meteorologic states and changes or disturbances on mucous membranes of the upper respiratory tract, the nasal accessory and tympanic cavities, certain conditions seeming to favor the development and spread of infections, others producing simple irritations, etc., and I have been impressed in numerous instances throughout the course of the recent epidemic by the striking similarity of conditions, except in degree. And then when one takes into account the fact that there has been a worldwide abnormal meteorologic state throughout the past two or three years, according to government and other scientific reports (with which, however, I confess but limited acquaintance), it seems to me in nowise unreasonable to conclude that there was probably brought into action such combined disturbances—atmospheric, electric, chemic, whatever it may be—as would so increase the virulence of bacterial organisms and so reduce the properties of human resistance that the pandemic was made possible.

To summarize briefly: considering the various phenomena observed during the recent epidemic in those who suffered the most virulent infections, and in others who, during the same period, exhibited characteristics similar in most respects, though milder in degree; the precipitate character of the epidemic's invasion in each locality, apparently precluding the possibility in numerous instances of contagion through personal contact one with another; the presence of all the usual bacteria common in normal times to infections of the upper respiratory tract, and the variableness of the predominant organism, not only in different

localities but in different individuals; the fact that meteorologic conditions over practically the whole earth have shown unusual disturbances during the past two or three years, and particularly during the past year; and that all of these seem to be so intimately associated, meteorologic influences may have played an important rôle as a causative factor in the great pandemic, and deserving of more careful consideration and investigation.



## CLINIC OF DR. MAURICE OSTHEIMER

### UNIVERSITY HOSPITAL

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#### INFLUENZA IN CHILDREN

THE most striking fact in the recent epidemic of influenza, in so far as it concerned children, was the uniform drowsiness, even stupor, observed in every instance. Even before taking the child's temperature the mother or the nurse noticed not only the flush due to the fever but also a marked drowsiness, almost a "doped" condition, in practically every case. Upon entering the hospital ward in which these cases were treated it was not difficult to pick out new cases of influenza, since these children lay quietly, often with their bottles but half full, apparently objecting to disturbance of any kind. In the uncomplicated cases the drowsiness and fever persisted for three to five days, usually less than a week, with some cough developing during this time in more than half of the children.

It fell to my lot to observe about 80 such children; in 48 of these only were there sufficient data for tabulation. This included 8 patients who were under treatment in the Children's Ward of the University Hospital, 3 of whom died of bronchopneumonia. These were the only fatalities among the 80 cases, a mortality rate of about 4 per cent. For the privilege of reporting these cases my thanks are due to Dr. J. P. Crozer Griffith and Dr. Howard Childs Carpenter.

A study of these 48 cases (Table I) shows that the sexes were almost equally divided, 25 girls and 23 boys, as was to be expected in an epidemic which was so wide-spread. Their ages varied from three months to twelve years (Table II). There were 18 under two years of age; 13 between two and five years; and 17 from six to twelve years of age. The highest temperature re-

TABLE I.—INFLUENZA CASES

	Sex.	Age.	Highest temperature, degrees F.	Duration of fever, days.	Drowsiness.	Loss of appetite.	Bronchitis.	Constipation.	Vomiting.	Pneumonia.	Aches and pains.	Abdominal tenderness.	Sore throat.	Termination.	Complications.
1	M.	3m.	101	3	++	++	+	++	+	++	+	+	+	R.	
2	F.	7m.	102	3	++	++	+	++	+	++	+	+	+	R.	
3	F.	9m.	102	3	++	++	+	++	+	++	+	+	+	R.	
4	F.	18m.	103	5	++	++	+	++	+	++	+	+	+	R.	
5	M.	3y.	102	3	++	++	+	++	+	++	+	+	+	R.	
6	F.	6y.	101	3	++	++	+	++	+	++	+	+	+	R.	
7	F.	7y.	100	5	++	++	+	++	+	++	+	+	+	R.	
8	F.	3y.	103	3	++	++	+	++	+	++	+	+	+	R.	Cervical abscess.
9	F.	6m.	104	28	++	++	+	++	+	++	+	+	+	R.	
10	F.	11y.	102	3	++	++	+	++	+	++	+	+	+	R.	
11	F.	3y.	101	3	++	++	+	++	+	++	+	+	+	R.	
12	M.	5y.	104	8	++	++	+	++	+	++	+	+	+	R.	
13	M.	7y.	103	5	++	++	+	++	+	++	+	+	+	R.	Epistaxis.
14	M.	12y.	102	3	++	++	+	++	+	++	+	+	+	R.	
15	M.	12y.	104	5	++	++	+	++	+	++	+	+	+	R.	Persistent sore throat.
16	F.	2y.	104	8	++	++	+	++	+	++	+	+	+	R.	
17	M.	6y.	103	9	++	++	+	++	+	++	+	+	+	R.	
18	F.	7y.	104	7	++	++	+	++	+	++	+	+	+	R.	
19	M.	8y.	103	9	++	++	+	++	+	++	+	+	+	R.	
20	M.	8y.	105	8	++	++	+	++	+	++	+	+	+	R.	
21	F.	2y.	105	8	++	++	+	++	+	++	+	+	+	R.	
22	F.	7y.	102	18	++	++	+	++	+	++	+	+	+	R.	
23	F.	4y.	104	10	++	++	+	++	+	++	+	+	+	D.	

[illegible]

corded varied from 100° to 105° F. (Table III). The number of days which the fever lasted also varied (Table IV). In sixteen cases, 33 per cent., the fever was present only three days; and in

TABLE II.—AGES

Months.	Cases.	
3.....	2	} 18 cases under two years.
6.....	3	
7.....	1	
9.....	3	
10.....	1	
11.....	1	
15.....	1	
18.....	4	
20.....	2	
Years.	Cases.	
2.....	2	} 13 cases between two and five years.
3.....	6	
4.....	4	
5.....	1	
6.....	4	
7.....	5	} 17 cases from six to twelve years.
8.....	3	
9.....	0	
10.....	2	
11.....	1	
12.....	2	

24 cases, 50 per cent., it lasted three to five days. Bronchitis and bronchopneumonia occurred in 43 cases, 89 per cent.; pneumonia, in 14 cases, 3 per cent. (with the 3 deaths already noted, a mortality of 21 per cent.); and bronchitis, in 29 cases, 60 per cent.

TABLE III.—HIGHEST TEMPERATURES

Fahr.	
100° in	1 case.
101° in	7 cases
102° in	14 "
103° in	10 "
104° in	12 "
105° in	4 "

The two common symptoms noted in every case, regardless of age, were the fever and the marked drowsiness, almost a stupor

in some cases, present even in the infants of three months. The symptom next most common was loss of appetite, noted in 33 children, 69 per cent. This symptom varied markedly among the children; some of the older children continued to eat well in spite of the evidences of toxemia. Other symptoms noted were constipation in 23 children, 48 per cent.; and vomiting in 17 children, 35 per cent. The aches and pains so commonly described by adults with influenza were present in only 13 children, 27 per cent., among the larger children, as was to be expected. Abdominal tenderness was noticed in 7 children only, 15 per

TABLE IV.—DURATION OF FEVER

		Pneumonia.	Death.
3 days in 16 cases		—	—
4 " 2 "		—	—
5 " 6 "		—	—
6 " 2 "		—	—
7 " 2 "		—	—
8 " 5 "		2	—
9 " 2 "		—	—
10 " 3 "		2	—
13 " 1 case		1	—
14 " 1 "		1	—
16 " 2 cases		2	—
18 " 3 "		3	1
19 " 1 case		1	1
24 " 1 "		1	1
28 " 1 "		1	—
—		—	—
48		14	3

cent. Sore throat also was a complaint in 7 of the children, 15 per cent. The complications which occurred were severe epis-taxis, severe and persistent sore throat, convulsions, asthma with persistent cough, cervical abscess, and a scarlatiniform eruption limited to the trunk, noted in one case each.

There were but 3 deaths, and these were due to broncho-pneumonia, as already stated. Almost every child affected by influenza lived in a family in which other cases of influenza had already developed. Most of the infants showed, besides the drowsiness and the fever, a distinct inclination to lie quietly, sleeping most of the time, without caring for much food. A few

ounces from each bottle, or even less at times, sufficed. But all the children, even the infants, took water freely.

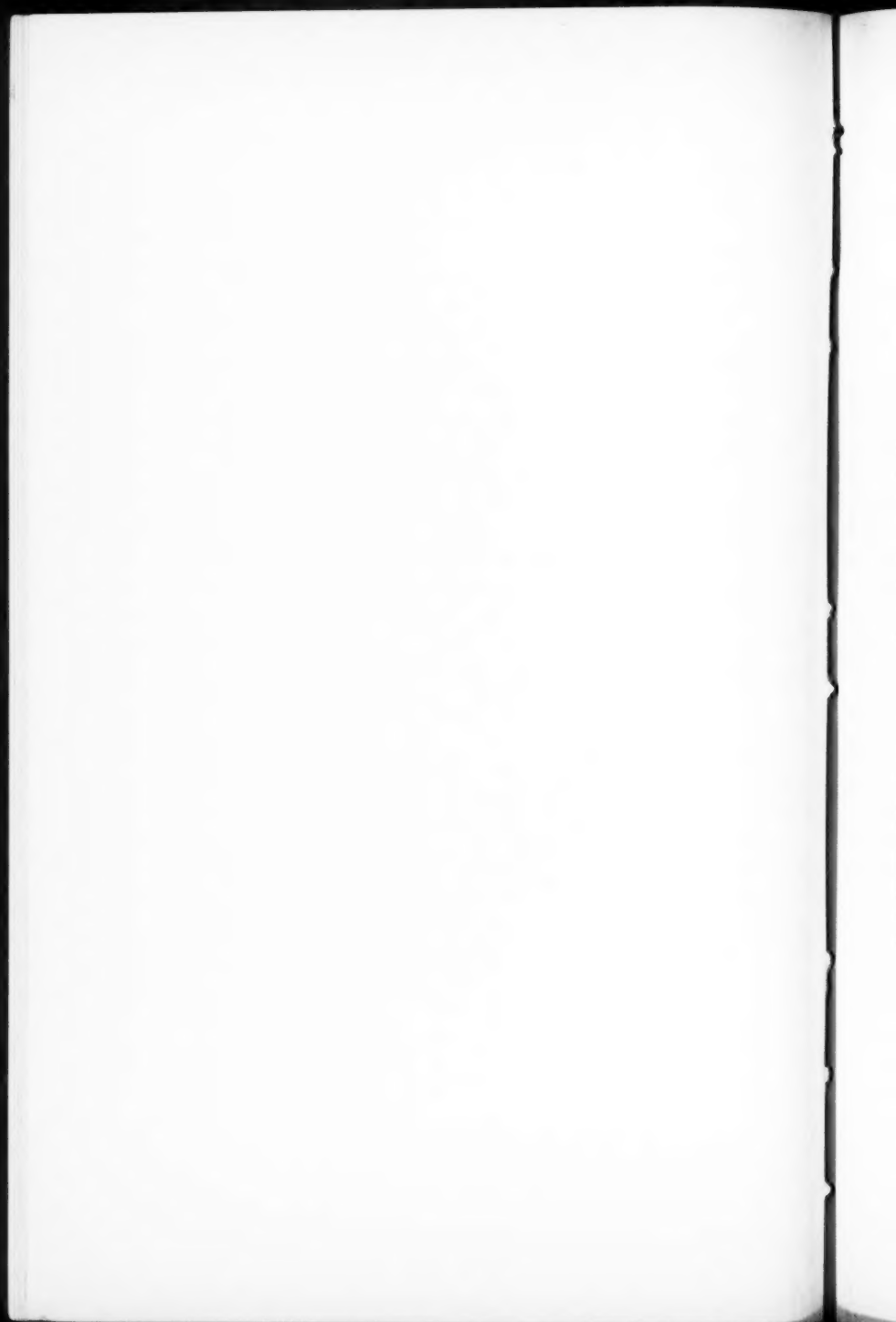
The routine treatment consisted of plenty of air, even keeping the child out of doors on sunny days, and always keeping windows widely open. On cool or damp days enough covers were used to prevent chilling the patient. A few children really needed hot-water bags under the covers; but the great majority did well in the cold air, without extra warmth. Drinking water was encouraged; a half cup to a whole glass of water was often given every hour or every two hours; to infants frequently in the bottle.

The only drug used was magnesium sulphate, in doses varying from 1 to 4 level teaspoonfuls, diluted in 3, 4, or 5 teaspoonfuls of water, given every morning, as long as the fever lasted. To those few children in whom vomiting occurred lime- and cinnamon-water, half and half of each, were given, in teaspoonful doses every half-hour, for eight or ten doses; followed by the salts, or by citrate of magnesia in doses of  $\frac{1}{2}$  glass, repeated in three or four hours when necessary. For the hard cough so common at the onset of the bronchitis or pneumonia 5 drops of syrup or wine of ipecac were given every two hours, in 2 teaspoonfuls of water, for five or six doses, until the cough became looser. Just as soon as the fever reached 99° F. or less a strong bitter tonic was begun, such as tincture of nux vomica, 5 to 7 drops; bicarbonate of soda, 5 to 7 grains; compound tincture of gentian, 20 drops, with enough water to make a teaspoonful, given before each meal. When the cough persisted, with expectoration, syrup of the iodid of iron,  $\frac{1}{2}$  teaspoonful well diluted in  $\frac{1}{4}$  glass of water, was kept up after meals, for some weeks.

While it was difficult to keep some of these children quietly in bed after the fever had disappeared, efforts were made to keep them in bed, allowing them to sit up in bed once daily for a short time, supported by pillows at first; later, twice a day; then without the pillows; and then only to try sitting up in a chair until tired. Thus convalescence was allowed to proceed very gradually, because a few of the earliest cases came down with a relapse after being up too soon, or after moving about and doing

too much the first day they got out of bed. Most of these children did not go about until the second week, and some not until the third week.

The coal-tar products and Dover's powder, etc., used at first, proved to be quite unnecessary, as so many of our physicians found true among adults also. Far better and quicker results were achieved without their use, especially among children, who respond so rapidly to even the slightest depressive action. Whisky, given occasionally before the doctor arrived, was found commonly to disagree with the child; several times vomiting and decided indigestion resulted from the whisky given before the physician reached the child.



## CLINIC OF DR. THOMAS McCRAE

### JEFFERSON MEDICAL COLLEGE

#### SCIATICA<sup>1</sup>

THE most common complaint with which patients come to us is pain or discomfort. We all recognize that this is not a disease, and that our efforts must be directed to determining the cause of the pain. We should never regard a diagnosis of pain alone as being a proper one. For example, to make a diagnosis of "head-ache" is evidently not even an approach to a proper understanding of the essential disorder. Yet, in the condition termed "sciatica" we too often rest satisfied with this diagnosis alone. A study of some of the patients to be shown should convince you that this is, in some instances at any rate, only scratching the surface.

There is one lesson difficult to learn, and when learned often forgotten, which is, that the seat of complaint of pain by the patient and the situation of the disease causing the pain are often distant from each other. Many examples will occur to you at once: A child with hip-joint disease complains of pain in the knee, a man with chronic appendicitis has gastric symptoms, prostatitis causes pain referred to the kidney, and so on. Therefore, when a patient comes complaining of pain it is wise to remember that the cause may be elsewhere. As you go on in medicine you will see many examples of the errors due to this fact being forgotten. This does not mean that it is not important to determine whether the pain has a local cause. It is wise to determine this early in the examination.

When pain is due to disease elsewhere there must be some association between the part where the pain is felt and its real

<sup>1</sup> This represents a combination of material from several clinics at the Jefferson and Pennsylvania Hospitals.

but distantly situated cause. This applies particularly to pain which is felt on the surface of the body. If you know anatomy thoroughly it is easier for you to trace the possible associations and hence determine the possible causes. If you do not know your anatomy, it will be a hopeless puzzle. This is another proof of the value of learning anatomy thoroughly. It would be well if this could be emphasized in the mind of the student when he is studying anatomy.

It is natural that pain referred to definite nerves should often be wrongly interpreted. The pain is so definite in its situation that a local condition in the nerve itself seems almost inevitable. Of course, there may be some change in the nerve, but this is not necessarily a primary process there. The condition known as sciatica illustrates this particularly well. The word is used to designate pain referred to the sciatic nerve. As an example of terms which mean nothing you will sometimes hear the expression "sciatic rheumatism" employed. This has nothing to do with rheumatic fever, but is an illustration of the use of the word "rheumatism" as meaning pain.

Let us go over the history and examine the first patient.

**History.**—CASE I.—P. J., aged forty-one, was admitted to the Pennsylvania Hospital on November 11, 1918, complaining of pain in the right hip, extending down the leg, and in the back.

**Past History.**—He had typhoid fever many years ago and this was the only acute illness he has had. There is no history of cough or shortness of breath. His appetite and digestion have been good; the bowels are regular and there is no history of any urinary trouble. The patient has three children, all of whom are well, and his wife has not had any miscarriages. There is an indefinite history of a luetic infection many years ago. He has used alcohol moderately.

**Present Illness.**—This began in the winter of 1917-18, but the exact date he cannot give. He states that he began to suffer with pain in the region of the right hip-joint, which gradually extended down the leg. The pain has grown worse, and in the last few weeks it has become practically continuous day and night. He has not been able to use the right leg and the pain has

interfered markedly with his sleeping. There has also been pain in the lower right back in the lumbar and sacral regions, but this has been less severe than the pain in the leg. As a result he has been much upset generally and has lost some weight.

*Examination.*—The temperature, pulse, and respirations are normal. The examination of the head, thorax, and abdomen shows nothing abnormal except chronic disease of the tonsils. There is no leukocytosis and the urine is clear. Rectal examination is negative. The left leg is perfectly normal. You can see that the right leg shows a great deal of discoloration from counter-irritation and many scars from wet cupping and the use of the cautery. He complains of pain throughout the whole leg, and on handling and movement the leg is painful everywhere. The patient indicates the most severe pain as situated over the sciatic notch and running down the back of the thigh. Below the knee he states that it is most severe over the anterior external surface of the leg and the dorsum of the foot. He keeps the right leg more or less in the position of flexion and slight abduction. There is no evidence of disturbance of the hip-joint. The thigh can be flexed on the body as long as the leg is flexed at the knee. If any attempt is made to extend the leg there is severe pain, as is very evident. There is more or less general tenderness over all the leg except on the front of the thigh. The special points of tenderness on pressure are over the sciatic notch, in the middle of the popliteal space, and opposite the head of the fibula. There is also very severe pain on pressure along the course of the anterior tibial nerve. The leg reflexes at present are, if anything, rather exaggerated. You can see that the tendo achillis reflex is well marked. There does not seem to be any marked disturbance of the sense of touch except that he is rather confused in distinguishing between a sharp and a dull object. There also seems to be some confusion in regard to the sensations of heat and cold. This is most marked over the dorsum of the foot and the outer side of the leg below the knee.

There is no doubt of the fact that the patient has severe

pain referred to the course of the sciatic nerve, and to this we give the name sciatica. Should we rest content with this as a diagnosis? Most certainly not, for this would be equivalent to a diagnosis of a symptom and not of a disease. Sometimes we have to rest content with such a diagnosis, but we should always regard it as a reflection on ourselves and leave nothing undone to get at the cause. As long as we are not satisfied with a diagnosis and have some "intelligent curiosity" to go deeper there is hope of learning the truth. Let me impress on you the point that in the great majority of cases termed "sciatica" we are dealing with a symptom of disease elsewhere. Do not accept this because I say so, but make up your minds from the evidence presented in the patients brought before you and those under your care after you graduate. In saying this do not understand me as expressing the opinion that there is no change in the sciatic nerve. There may be definite changes of an inflammatory nature. The important point to learn is that these are rarely primary. You may ask where the line is to be drawn in excluding some grade, perhaps slight, of neuritis. For practical purposes you do not need to draw any line. In fact, there are transition forms between what may be called a neuritis and a neuralgia.

It is possible that this patient may be one of the rare examples of a true neuritis of the sciatic nerve. If this is the case we would expect to find a definite change in the reflexes, particularly in the tendo achillis reflex, which would be diminished or absent. In this patient you note that this reflex is increased. A marked grade of muscular atrophy or alteration in the electric reactions would also be in favor of a true neuritis.

Therefore we conclude that we are not dealing with a true neuritis, but with a condition in which a painful sciatic nerve is due to disease elsewhere. The scars over the course of the nerve are good evidence of the thoroughness with which local treatment in the form of counterirritation has been employed. Had the cause of the pain been in the nerve we should have expected some improvement, but the patient is very emphatic in his statement that any benefit was slight and only temporary.

He is the one who should know. Our next effort must be directed to deciding what and where this disease process is. If you will consider the anatomy of the sciatic nerve it may help us to state the possibilities. It is evident that there are many chances of the nerve being affected by disease elsewhere. Let us study some of these in the patient. We have already found that there is no evidence of any disease of the hip-joint. Examination of the sacro-iliac joints does not show anything to suggest disease there. Examination of the prostate is negative. One point in the history should suggest a possibility to us. He complains of pain in the lower back on the right side. This always demands a careful study of the spine.

When the patient stands, the examination of the spine shows signs of some change there. The muscles on the right side of the spine in the lumbar region are seen to stand out more prominently, and on palpation they are felt to be much more firmly contracted than on the other side. On testing the movements of the spine you can see that they are much decreased in certain directions. Movement is much restricted on bending forward, backward, and to the right. He has more movement on bending to the left. This is suggestive of some disease in the spine and is confirmed by the x-ray examination, the report of which is as follows: "There is no lesion found in the sacro-iliac regions. There are some areas of calcification, apparently united to the upper border of the third and fourth lumbar vertebræ on the right side; in each case extending upward and nearly meeting the lower border of the body above. Apparently a case of hypertrophic spondylitis" (Fig. 141).

With spondylitis it is common to find pain referred to the nerves which may be affected by the process. This may be by pressure or communicated inflammation. Therefore it would seem reasonable to conclude that in this patient the process in the lower spine is responsible for the sciatica.

Should we stop there? We have made an *anatomical* diagnosis of spondylitis, but it is evident that we should try and go further and determine the *etiology* of this if possible. In this let me suggest one disease to be excluded in such cases—*syphilis*. It is

a good rule to exclude syphilis in every obscure case of sciatica and perhaps also in cases that do not seem obscure. Syphilitic disease of the bones of the spine in the form of arthritis is not



Fig. 141.—The x-ray picture of the lower spine (Case I).

very rare. In this case there are no signs of syphilis and the Wassermann reaction is negative. Excluding this, we regard such a process in the spine as a form of arthritis deformans and

due to a focus of infection somewhere. The routine examination shows marked infection of the tonsils, and it seems a fair inference that his arthritis is due to this. (Later the tonsils were removed and pus was found in both of them.)

Let us go over our diagnosis again in its details:

1. We found a painful sciatic nerve, the condition known as sciatica or, as it is sometimes termed, "sciatic neuralgia."

2. Next we find a spondylitis, to which we regard the sciatica as secondary.

3. In the search for the cause of the spondylitis we find infection in both tonsils.

In the order of events we should say (1) chronic tonsillitis, (2) spondylitis with new bone formation and undoubtedly some inflammation of the soft structures, and (3) sciatica. It is quite evident that had we been content with the diagnosis of sciatica the real disease would not have been recognized. The bearing of this on treatment is evident.

The next case illustrates other possibilities in the search for the cause of the sciatica.

**CASE II.**—This patient is a male, aged forty-seven, who has a negative past history. He has always been active and has taken a great deal of exercise. There is no history of gonorrhea or lues.

*Present Illness.*—The onset of his trouble was about one year ago, when he had a fairly sharp attack of pain which began suddenly in the region of the right sciatic nerve. He kept about at first, but in three days he was compelled to go to bed, where he remained for a week. He then got up, but has had some trouble in the same region ever since. His work compels him to stand a good deal, and this usually aggravates the pain. In addition, he states that there has been a certain amount of numbness in the right foot. About a month ago he had what he terms a "lame back." This troubled him a good deal, particularly in stooping, but did not compel him to go to bed. Two weeks ago there was a very sudden return of the sciatica in such a severe form that it has been difficult for him to get about, as any attempt to walk causes severe distress. He has had a great deal of pain at night. On

questioning him carefully regarding the pain in the back, which he terms "lumbago," he tells us that he has had it a number of times, the first attack being perhaps twenty years ago. He states that it has never been severe enough to compel him to go to bed.

Let me digress for a few minutes to discuss the significance of this part of the history. The term "lumbago" is loosely used, and is generally employed to designate pain in the lower back. We should never be content to make such a diagnosis. It would be better to term it "pain in the back" rather than lumbago, because when you have given a name to anything you have to some extent disposed of it. Look on lumbago merely as a symptom and not as a disease, and do not rest until you have discovered what is causing the pain.

*Examination.*—You can see that the patient looks very well nourished and has a good color. There is no evidence of disease in the thoracic or abdominal viscera. He does not seem to have any particular difficulty in sitting down or getting up from the sitting position, but you observe that he becomes uneasy if he is kept standing for more than a short time and complains of pain which he refers to the region of the right sciatic nerve. He has severe pain when an attempt is made to flex the thigh on the trunk with the leg extended. On testing the movements there is no restriction at the right hip-joint, but on flexing the thigh to about right angles with the trunk a very definite click is heard, apparently in the region of the right sacro-iliac joint. Pressure over this joint does not seem to cause any discomfort. Forcible extension of the right thigh on the trunk causes severe pain referred to the sacro-iliac joint.

This seems to suggest very definitely some disease in the sacro-iliac joint. There are two methods by which we may be more certain about this. One is by an *x-ray* study and the other by the result of fixation of the joint. The report of the *x-ray* study by Dr. Manges is as follows: "There is apparently absorption of the cartilage in the sacro-iliac joint, so that bone approximates bone, especially in the upper half of the joint surface. There is no evidence of any disease in the lumbar vertebrae."

On rectal examination it was found that the prostate was considerably enlarged and somewhat tender. This naturally brought up the question as to whether the sciatica might not represent a reflex pain from the prostatitis. The therapeutic test was of considerable help in deciding this point. We fixed the sacro-iliac joint as far as possible by strapping and secured a very striking result. The patient states that within two days there was very marked relief and within a few days after this the pain had entirely disappeared. We kept up a certain amount of fixation of the sacro-iliac joint and as long as this was done the patient was comfortable. This result seemed to definitely exclude the possibility that the pain might have been referred from the prostate. We have given him directions to be very careful to avoid any sudden lifting or any movement which might strain the joint.

This brings before us an entirely different etiology for sciatica. In the previous case we tried to carry the etiology farther back than the anatomical lesion. Can we do so here? You remember that in his history he stated that there had been attacks of pain in the back at intervals for twenty years. Has this been due to the lesion in the sacro-iliac joint? It may be so, but other causes have to be considered. One of these is spondylitis, but if he had this for such a long period of years we would expect to find some changes in the bones. The x-ray report excludes these. A second possibility is that the prostatitis may be the cause. It is difficult to exclude this, but we would expect more symptoms to have appeared by this time if the prostatitis is of such long duration. He has no symptoms of a urinary or sexual character and, therefore, I am inclined to the view that the sacro-iliac trouble has been responsible. If so, the cause may have been an injury of the joint through some sudden strain. A process of infection may be responsible, but he does not show any evidence of this unless it be in the prostate.

It is not difficult to understand why disease of the sacro-iliac joint causes pain referred to the course of the sciatic nerve. The lumbosacral nerve passes over it and hence is easily affected by disease of the joint.

The next patient shows the same condition of sciatica, but with some additional features and another cause.

**CASE III.**—The patient, aged fifty-two, occupation farmer, comes complaining of pain associated with disturbed sensations in the left leg.

*Past History.*—The only acute disease was an attack of dysentery in youth. His appetite and digestion have been good, and the bowels regular. He has used tobacco heavily and alcohol moderately. There is no history of gonorrhea or lues. For some time past he has been troubled by urinary symptoms and has had a certain amount of difficulty in urinating, but this has never been very serious.

*Present Illness.*—The exact time of onset is difficult to determine, but the symptoms probably have been present for about three years. He first noticed a disturbed sensation, particularly in the lower left leg. This he describes as a burning sensation. Some time afterward he began to have pain in the lower left leg on walking or standing. He does not give a very definite account of the location of this in the early months, but it gradually extended higher up until for a year past it has been referred to the course of the sciatic nerve. At the same time that he has the feeling of pain he also has a sensation as if the whole leg was "dead." This disturbed sensation is more marked over the thigh than below the knee. He sticks to his description of severe pain and a sensation of "deadness," as he describes it, as co-existing in the leg. He also feels that there is some loss of power in the left leg, but he has always been able to get about, and has never had to go to bed with the condition. He has some difficulty in balancing on the left leg and states that he feels that it is "unreliable." The condition has disturbed him a good deal and has interfered with his sleep. As a result of this he has lost about 30 pounds.

*Examination.*—The patient's general condition is good and nothing abnormal is found about the head, the chest, or abdomen. The prostate is found to be considerably enlarged, very tender, and the secretion contains a large number of pus cells.

On walking, the patient shows some slight difference in the

action of the two legs. This is more marked when he attempts to walk upstairs. There is a slight difference in the size of the legs, the left being the smaller. The knee-jerks and tendo achillis reflexes are well marked and equal on the two sides. It is not possible to determine any definite alteration in sensation even over the areas where this is most marked according to his account.

Examination is absolutely negative with reference to any other cause for the sciatica. There is no evidence of any disease of the bones, of the spine or pelvis, or of the sacro-iliac and hip-joints by ordinary examination and by x-ray studies. The disease of the prostate seems to be the only explanation. Treatment for this was begun promptly, and the patient thinks that his condition is improving, but as yet not sufficient time has elapsed to give any opinion regarding the ultimate outcome.

One feature in the history deserves emphasis, and this is the fact that the various sensory disturbances antedated the appearance of the pain in the sciatic nerve. It is also worth noting that these sensory disturbances are entirely subjective. We are not able to find any evidence of disturbed sensation by the usual tests.

In this case the only etiologic factor which we can find is the chronic prostatitis. You note in the history that he had a good deal of indefinite sensory disturbance for some time before the onset of the sciatica. The pain has apparently not been as severe in this patient as in the two preceding patients. It has seemed to me that this is true in the cases of sciatica secondary to prostatic disease that I have seen. The cause of pain referred to the sciatic nerve may be (1) from pressure on the nerve, especially seen in some cases of carcinoma of the prostate, and (2) from the association of the roots of the pudic nerve with the sacral plexus.

**Discussion.**—You may ask very properly as to the relative frequency of these causes. A most interesting study by Rogers<sup>1</sup> is worth quoting in this connection. He studied a series of 50 cases. In 49 there was a lesion of the lower spine, lumbo-

<sup>1</sup> Jour. Amer. Med. Assoc., 1917, lxviii, 425.

sacro-iliac joints, in 1 there was carcinoma of the prostate; 8 of the cases had vertebral arthritis and 4 had tuberculous disease. In this series disease of the sacro-iliac joint was the most frequent cause, and Rogers gives the excellent suggestion that the pain produced by the endeavor to flex the straight leg on the trunk is not due to stretching of the sciatic nerve, but to the condition in the sacro-iliac joint. This is a point well worth having in mind in the study of your patients.

There is also the question as to how often the pain is due to a true sciatic neuritis. Judging from my own experience I should say extremely rarely. The more thoroughly you study your patients, the rarer such cases will be, and the chances are that many of you will never see one. You will see the statement that sciatica occurs in diabetes mellitus and gout. This cannot be denied, but the cases occurring in diabetes must be very rare. If it occurs in a patient said to have gout, make a very careful study to be sure that you are not dealing with arthritis deformans, possibly with spondylitis. As to the explanation that sciatica is due to the so-called "rheumatic diathesis," do not give it a place in your memories. The term has no definite meaning and usually covers ignorance. It sounds plausible, but may be dismissed.

**Other Causes.**—There are additional causes of sciatica which should be kept in mind. They are rarer than those demonstrated in these patients.

(1) *Hip-joint Disease.*—This should not cause difficulty if a proper examination of the joint is made. The use of the *x*-rays is of great value in recognizing it.

(2) *Lesion of the Cauda Equina.*—The signs of this vary with the extent of the process, but unless the lesion is low there will be paralysis, and in all cases there is likely to be an area of anesthesia. The sensory and motor symptoms are distributed in root areas and not according to the peripheral nerves. Examination of the condition of the sphincters of the bladder and rectum is important.

(3) *New growth or tuberculosis* of the bones of the lower spine or pelvis. These may cause symptoms like those produced

by changes in the bones due to arthritis. The x-ray examination should give definite information.

(4) *Varicose or Phlebogenic Sciatica*.—This is a very rare form, but with features which enable it to be recognized quite readily if you know about it—which is true of many things in medicine. There is severe pain referred to the sciatic nerve, sometimes accompanied by painful cramps in the gluteal muscles. When the patient stands or walks slowly the pain is severe, and is usually most marked in the popliteal space and below the head of the fibula. When he lies down or exercises the legs vigorously the pain disappears. Examination with the patient lying down may not show any abnormality, but there is pain and tenderness when he is examined in a standing posture. This form is due to varicose veins within the sheath of the sciatic nerve. Occasionally there is varicosity of the superficial veins and slight puffiness in the popliteal space with it. The treatment is mechanical by the use of an elastic support.

(5) *Pelvic Disease*.—It can be readily understood that any condition in the pelvis which causes pressure on the nerve may give rise to sciatica. It has resulted in parturition from the pressure of the child's head. Pressure from a mass due to pelvic inflammatory disease or from a malignant growth may be responsible. Hence the necessity of always making a rectal examination and a vaginal examination in women.

(6) *Psoas abscess* is a rare cause.

(7) *Syphilis*.—This should be considered in every obscure case. Some writers have discussed it as a primary syphilitic neuritis, and this is possible in the form of a sclerogummatous neuritis. But before accepting such a diagnosis careful study should be made to exclude other possibilities, not forgetting that syphilis often causes spondylitis. A syphilitic meningitis or periostitis may be responsible. In going over some of the reports in the literature of cases regarded as syphilitic one is impressed by the absence of any records which point to a proper study of the spinal and pelvic bones and joints. There do not seem to be any features by which we can recognize a sciatica due to syphilis other than by the serologic and therapeutic tests.

Some place emphasis on the pain being particularly severe at night, but this is true of many cases which are certainly not due to lues.

(8) *Gonorrheal Sciatica*.—This is described in the literature, but must be very rare. One would suspect that in such cases there had been an arthritis to which the sciatica was secondary.

**Diagnosis.**—It may be said that in the great majority of cases the recognition of sciatica is not difficult. The features are usually quite characteristic. The next step is to decide whether the pain is due to a true neuritis or to a neuralgic sciatica. The former is extremely rare, and the important points have been mentioned. Observation of the tendo achillis reflex is especially important in this. With a sciatic neuralgia the next step is to decide as to the cause. This involves a thorough study of the lower spine, the pelvic joints and bones, and the pelvic organs. Determine first if there is any evidence of disease of the lower spine, then examine the sacro-iliac joint and the hip-joint. In studying these areas the information from the x-ray plates is of the greatest aid. Then examine the pelvis with reference to its organs. If no evidence of disease is found in any, then consider the rarer causes mentioned above, not forgetting syphilis.

Further, we should make the effort to go still farther back and determine the cause of the lesion. This applies especially to arthritis of the spine as was shown in Case I. I should not like you to go away with the idea that the diagnosis of the cause is always as easy as it has been in these patients. That would be too much to expect, and you will have patients in whom the cause is very obscure and in whom the search seems hopeless. Still something is gained if you recognize this and feel that the cause is still to be found. This is better than going no farther than making a diagnosis of sciatica only.

The errors that are made in the recognition of sciatica are usually due to a careless examination. In some cases the pains of tabes have been regarded as those of sciatica. Osteomyelitis has caused pain which has led to a mistaken diagnosis. The pain due to flat-foot or varicose veins should not cause error.

In one case known to me the pain associated with intermittent claudication had been regarded as sciatica. Anatomic abnormalities in the lower vertebræ and especially in the transverse processes may cause pain which simulates sciatica.

**Prognosis.**—The same warning may be given here. Do not expect to secure in every case as rapid results as are shown in Cases I and II. If you do, some sad disappointments are in store for you. The first patient entered the hospital almost unable to get about and having constant severe pain except when under the influence of sedatives. Within a week he was much improved and within three weeks was waiting on the other patients as a volunteer orderly. The second patient had relief in two days and within a week stated that he was perfectly well. He was free of pain, but the lesion in the sacro-iliac joint was still present.

It is evident that the prognosis depends very largely on your ability to influence the cause. But two of the common causes—spondylitis and sacro-iliac joint disease—are often very difficult to influence, and hence the need for caution. We are fortunate in these patients, but the next one may be more difficult to help. Therefore, always be guarded and explain to the patient that the outlook may depend on factors which are sometimes difficult to remedy. Your knowledge of the etiology is of great value in prognosis and treatment, but it also aids you to realize that the course may be protracted. It explains the cases of chronic sciatica in which the pain recurs over long periods. The other pains due to spondylitis and sacro-iliac joint disease have the same character. However, it is fair to say that in general an exact diagnosis is of help in prognosis—whether this be favorable or unfavorable as regards the probable duration of the pain.

**Treatment.**—It is evident that no proper treatment can be given for sciatica which is not based on an etiologic diagnosis. To relieve the symptom of pain is essential, and to this extent a symptomatic treatment is proper. The management of the cause will be very different, as is shown by the patients you have seen. In one, fixation of the spine with removal of the infected tonsils; in the second, fixation of the sacro-iliac joint; in the third, treatment of the prostatitis was required. The man who

insists on proper diagnosis as an essential for proper treatment is often termed a "therapeutic nihilist," exactly why it is difficult to discover. But surely this probalem of sciatica illustrates the importance of proper diagnosis. There are articles on the treatment of sciatica in which there is not a word of reference to the etiology and to the need of attending to the cause. They deal only with the relief of pain.

However, in the majority of cases it is necessary to relieve the pain. The first essential in this is *rest*. Let it be emphasized that this should be complete and for some time, the duration to be determined by the progress. The patient should be in bed and move as little as possible. He should not get up to go to stool, but use the bed-pan. Every effort should be made to secure comfort. Sometimes a splint for the leg is advisable, but usually proper support in the easiest position can be secured by pillows. If there is disease of the spine, it may be well to see that the back is properly supported. If there is marked improvement in a short time, as often happens, you may have trouble in keeping the patient quiet. Warning them that an hour up and about may undo the effect of a week's rest may have some effect. The length of the period of rest has to depend somewhat on how successful you are in discovering and treating the cause. Thus in the second case the period of rest was only two days and the patient was going about within a week. Such a result is exceptional. If you cannot find any cause, rest for some weeks is usually indicated.

There are a number of drugs which may be given for the relief of the pain. The salicylates are generally of value, and my preference is for the sodium salicylate in doses of 15 grains four times a day. The dosage may soon be reduced if the result is satisfactory. It is often of advantage to combine sodium bromid (10 grains) with the salicylate. Acetyl-salicylic acid may be given up to 30 or 40 grains a day for a short period. The combination of phenacetin and salol (5 grains each) three times a day is sometimes useful. Other drugs of the coal-tar class may be given, and sometimes a combination of small doses of several of them is more effectual than a larger dose of one. It is well to try and avoid the use of opium or its derivatives. Morphine

should be given only if other measures fail to give relief and the pain is very severe. Its administration should not be left to the patient or to a member of the family. If an opium derivative is necessary, codein ( $\frac{1}{2}$  to 1 grain) in combination with acetylsalicylic acid or phenacetin should be used.

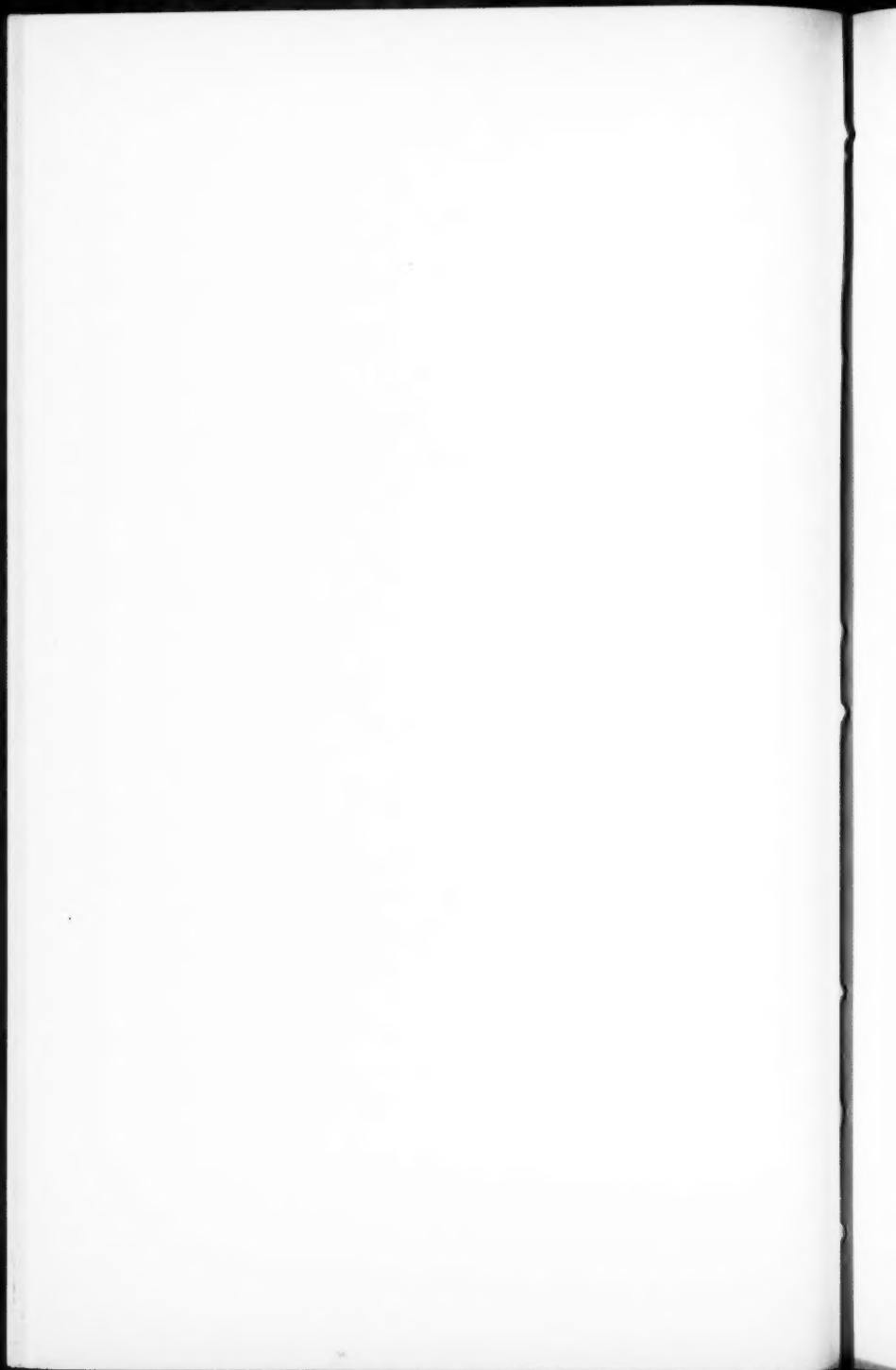
Local measures are often helpful, and the extent to which they may be employed was demonstrated by the scars on the first patient you saw. Counterirritation should be applied over the course of the nerve and may be given in various ways. The Paquelin cautery takes first place, but does not need to be applied very vigorously. It is not necessary to do more than touch the skin superficially. The use of blisters and the application of mustard may give relief, but always keep in mind that these measures should be used in moderation, as the patient may have to lie in bed for some time.

Acupuncture has been employed and sometimes gives relief. Injections of sterile water or weak cocain solutions may give temporary relief, but there is danger of paralysis, which has resulted in some cases. The injection of solutions which may injure the nerve is not advisable. The operation of stretching the nerve has very properly gone out of use.

In chronic cases it is natural that a variety of treatments should be tried. Hydrotherapy is sometimes of value, but more often gives only temporary relief. Both it and massage are contraindicated when the condition is acute. Electricity sometimes gives temporary relief to the pain, but in general its use is disappointing.

General measures should be employed when indicated. Iron and arsenic are essential if anemia is present. A syphilitic sciatica requires the usual specific treatment. A special diet is only indicated in cases of sciatica due to gout or diabetes. The advice to cut out red meats from the diet does not seem to have any proper basis, unless for some other reason you wish to reduce the protein intake.

Essentially the treatment of sciatica should be based on the removal or correction of the cause. Till this can be accomplished we should endeavor to relieve the pain, but this is to be regarded as only palliative and not curative.



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**INTRASPINAL THERAPY IN SYPHILIS**

THE medical profession is essentially conservative in placing the stamp of its approval upon new therapeutic procedures. No fault can be taken with this attitude, as medical history is replete with instances of new methods of treatment loudly heralded by their advocates and confirmed by others as important advances in the healing art, which were, after maturer trial, consigned to well-deserved oblivion. On the other hand, genuine and at times important contributions to our therapeutic knowledge are often viewed with obstinate incredulity for too long a period. One need only recall to mind the heated controversies of Harvey, Jenner, Pasteur, and Lister, and the difficulties they had to overcome to persuade a skeptical profession of the truth of their theses.

There are many physicians today who are still unconvinced of the value of intraspinal medication in syphilis of the nervous system. A sufficient experience has, however, accumulated to enable one to definitely state that this method of treatment must be regarded as a valuable, if not indispensable, addition to our therapeutic procedure.

We have been employing intraspinal treatment for several years. Our earlier results were not particularly encouraging, and we abandoned this treatment for some time, but results which were achieved from time to time have convinced us of the indispensability of this method of treatment in appropriate cases.

We present the history of a group of cases in which intraspinal treatment was employed. The facts speak for themselves.

CASE I.—J. C., twenty-nine years of age, was infected in 1908. He took mercurial pills steadily for three years under the

advice of his family physician. One year later his Wassermann was said to be negative. With the consent of his physician he married. He came under the care of one of the essayists in March, 1918. Patient is a vigorous specimen of manhood with well-developed musculature and weighing 180 pounds. He recently had a Wassermann test made both before and after a salvarsan injection, and the tests were reported as negative. He has two children, who are robust and healthy in appearance.

Examination revealed a sluggish reaction of the left pupil and impaired patellar reflexes, which later were entirely lost. No Romberg; gait normal. Patient complained of being "nervous." The blood Wassermann, on examination by Dr. John A. Kolmer, was  $+$ <sup>4</sup>, with three different antigens. (It was reported negative by a technician in another city but a short time before.) The spinal fluid findings were as follows: cells 50: W. R., cholesterinized antigen  $+$ <sup>1</sup>, alcoholic extract syphilitic liver  $+$ <sup>1</sup>, acetone insoluble lipoids  $+$ <sup>4</sup>. (A moderately positive Wassermann.) Protein showed a trace both by the Pandy and Noguchi tests. The colloidal gold test exhibited a suggestive luetic zone curve—0011110000.

The patient received on 4/17/18 0.6 gram arsphenamine intravenously, followed by the intraspinal injection of auto-arsphenamized serum, to which  $\frac{1}{4}$  milligram of arsphenamine was added. On 5/1/18 he was given 0.6 gram of arsphenamine, followed by spinal drainage. The spinal fluid at this time showed a marked improvement over the test made two weeks previously: spinal Wassermann  $+$ <sup>2</sup> —  $+$ <sup>1</sup> with 1 c.c. of fluid. The cells were reduced to 21. Protein, a trace. Gold test, 0011000000. 5/22/18 0.6 gram arsphenamine intravenously, followed by spinal drainage, 24 c.c. being withdrawn. On 5/24/18 the blood Wassermann was  $+$ <sup>3</sup>  $+$ <sup>1</sup>  $+$ <sup>2</sup> (a moderate positive). The patient reported again 6/18/18, at which time the pupils responded to light with greater alacrity. The right pupil was normal; the left a little less responsive; 6/18/18 spinal fluid W. R. negative; cells 9; gold test negative. The blood Wassermann was still moderately positive,  $+$ <sup>3</sup>  $+$ <sup>2</sup>  $+$ <sup>2</sup>. The patient had lost his nervous feeling and declared himself to feel normal in every respect. In

this case the pathologic spinal findings were restored to normal within a short period of time, although the blood Wassermann was practically unchanged. To be sure, the spinal fluid must be further studied to make sure that the findings do not deteriorate. The patient is still under treatment.

CASE II.—C. S., age thirty. Infection took place two years ago. The patient was treated by intravenous injections of arsphenamine. He had two negative blood tests at intervals of two months, several months prior to the patient's visit to us. The spinal Wassermann was + + + +, with all of the antigens employed. In March, 1918, he developed a diplopia. The eyes had never exhibited evidence of inflammatory trouble, but since the fall of 1916 his vision progressively failed. In May, 1917, Dr. T. B. Holloway examined his eyes and found "the vision in the right eye 6/60, with  $-1.00 = -.75$  cyl. ax. 15, and the vision in the left eye 6/21, with  $-1.25$  S. =  $-.50$  cyl. ax. 90. The pupils measured 6 mm. and were slightly irregular in contour. They reacted slightly to accommodation and convergence, but not to light. There was a partial paralysis of the right sixth nerve, with practically complete paralysis of the left sixth, together with partial paralysis of the left third nerve. Each disk was markedly atrophic."

The patient was given twenty-four intravenous injections of arsphenamine with eighteen Swift-Ellis spinal injections, reinforced by the addition of  $\frac{1}{2}$  mg. of arsphenamine.

After this treatment the spinal Wassermann became negative. Dr. Holloway reports that "it is interesting to note that while the vision was progressively decreasing prior to the time the patient came under observation and treatment, that since the latter was instituted, the progress has apparently been checked. The optic atrophy has not shown any change, but a slight reaction to light has developed in the right eye during the course of treatment. There has been no decided improvement in the condition of the ocular muscles."

The interesting features of this case of *double optic atrophy* with *paresis of the ocular muscles* are the presence of a negative blood Wassermann, with a strongly positive spinal Wassermann,

and the apparent checking of the atrophy by intensive intravenous and intraspinal treatment. There can be little doubt that this patient's vision could have been saved if a diagnostic spinal puncture had been made earlier and appropriate treatment instituted.

CASE III.—S., age twenty-three. Patient developed a chancre in December, 1917, followed by a secondary eruption and mucous patches.

In March, 1918, the patient developed retinal hemorrhages. His nervous system, heart, and lungs were normal as far as any discoverable evidence could be elicited. The blood Wassermann was weakly positive. The spinal fluid findings were as follows: cells 10, Noguchi globulin test weakly positive; spinal Wassermann weakly positive. He received from us twelve intravenous injections of arsphenamine in 0.4 to 0.6 gram doses without any material improvement of the retinal hemorrhages. On 5/24/18 he received a Swift-Ellis spinal treatment, reinforced by  $\frac{1}{4}$  mg. of arsphenamine. The blood Wassermann became negative; the spinal Wassermann was weakly positive. On 6/24/18 he again received an intraspinal treatment containing  $\frac{1}{3}$  mg. arsphenamine. The spinal fluid then became Wassermann negative. Dr. Luther C. Peter, who examined the patient's eyes, reported that after the second intraspinal treatment the retinal hemorrhages exhibited a marked improvement.

Ordinarily retinal hemorrhage is favorably influenced by intravenous administration of arsphenamine, but in this patient much better results were obtained by the conjoined use of intraspinal treatments.

CASE IV.—B. K., age forty, came to see us complaining of pains in the back, legs, and loss of sensation in both feet. Duration two years. There was no history of a syphilitic infection; the wife and children were Wassermann negative. The patient's blood Wassermann was + + + +. Examination revealed Argyll-Robertson pupils, marked Romberg, and absent knee-jerks. The patient received five intravenous injections of arsphenamin, with very little improvement. The spinal fluid was + + + +, with three different antigens. The cells were not counted. The

Noguchi globulin test strongly positive. Diagnosis, tabes dorsalis. The patient received five intraspinal arsphenamine treatments (Swift-Ellis-Ogilvie technic) at intervals of two weeks. After this treatment the globulin test became negative, but the blood and spinal Wassermanns remained++++. The pains in the back and legs practically disappeared. His gait is improved as regards stability of footing and he no longer suffers formication in the feet.

CASE V.—M. B., age forty-three. Patient had initial lesion fifteen years ago, followed by secondaries. He was treated with mercury by mouth for one year. He complains of weakness in the legs and feet, shooting pains, and difficulty in walking. Duration two years.

The patient presents Argyll-Robertson pupils, marked Romberg, and absent knee-jerks. No changes in sensation. The peripheral vessels show marked arteriosclerosis. Diagnosis, tabes dorsalis. The blood Wassermann is++++; the spinal Wassermann is++++. Cells 30. Globulin positive. The patient received twelve intravenous injections of arsphenamine and ten intraspinal treatments. He was then allowed a rest of several months. On his return the blood Wassermann was +<sup>1</sup>, with a cholesterinized antigen, and negative with extract syph. liver and acetone insoluble lipoids. The spinal Wassermann was +<sup>4</sup>, with cholesterinized antigen, +<sup>1</sup> with alcoholic extract syphilitic liver, and +<sup>8</sup> with acetone insoluble lipoids. The globulin test (Pandy and Noguchi) was negative and the cells had been reduced to 3 per cubic centimeter. The patient's pains are decidedly less and his walking is distinctly improved. He later received three more intravenous and intraspinal treatments. The blood Wassermann became negative, but the spinal Wassermann relapsed to a++++. The globulin was negative and the cells normal.

CASE VI.—O. C., age forty-three. No history of syphilitic infection. Patient complains of weakness in left arm and leg, and of double vision. Duration of symptoms one year. The pupils are small and irregular and react but slightly to light. The left knee-jerk is increased; the right is normal. Left ankle

clonus present; right side absent, no Babinski. No changes of sensation. The blood Wassermann is + + + +; the spinal Wassermann is negative. Cells not counted. Globulin test positive. Diagnosis, syphilitic meningomyelitis (?). The patient received fourteen intravenous injections of arsphenamine. At the end of this course of treatment the blood Wassermann was still + + + +. The patient was then given reinforced Swift-Ellis spinal treatments; after this treatment the spinal Wassermann changed from negative to + + + +, apparently a provocative Wassermann. After the second intraspinal treatment the patient was so markedly improved that he resumed his work and wrote that he felt that he did not require further treatment.

CASE VII.—W. W. S., age forty-three. Infection in March, 1903. Had an initial lesion and a generalized eruption. Took mercury pills for a month or two. No further treatment until 1918. Sought medical advice in 1918 because of diplopia. The pupils are irregular and small. Right pupil does not react to light; the left sluggishly. There is a ptosis of the right upper eyelid. The patellar reflexes are greatly exaggerated. In the right leg the Babinski is plus; in the left leg, normal. The Romberg is pronounced; approximation of fingers with the eyes closed is poor. Gait is fairly good. Some vesical dribbling. The blood Wassermann is +<sup>4</sup>, the spinal Wassermann is likewise +<sup>4</sup>. There were but three cells to the cubic centimeter. There was an increased amount of globulin, and the Lange gold test showed a tabetic curve.

The patient has received seven intraspinal injections of arsphenaminized serum following intravenous injections upon the same or on the previous day. The patient is much stronger, he is steadier on his feet, and the co-ordination of his hands has improved. The diplopia and the ptosis have likewise improved greatly, although a trace of each remains. The blood and the spinal Wassermann both remain strongly positive.

CASE VIII.—J. L., age fifty-two. Infection dates back nineteen years. Local treatment applied to initial lesion. No internal treatment.

Present complaint is weakness in the legs and burning sensation in the feet. The patient likewise complains of pains in the stomach. Duration of these symptoms ten years.

Physical examination reveals an Argyll-Robertson pupil, absent knee-jerks, and extremely poor station. The blood Wassermann is  $+$ <sup>4</sup>; the spinal Wassermann is  $+$ <sup>4</sup>. The Pandy and Noguchi globulin tests are positive. The colloidal gold test is suggestive of the paretic curve. Cells not counted.

Patient received Swift-Ellis intraspinal treatments reinforced with  $\frac{1}{4}$  to  $\frac{3}{4}$  mg. of arsphenamine, at intervals of two to three weeks. The blood Wassermann and the spinal Wassermann are still strongly positive, but there is pronounced clinical improvement. The patient is stronger, steadier upon his feet, and walks almost normally.

CASE IX.—Age fifty-three. Initial lesion nine years ago. A secondary eruption was not observed. The patient received no internal treatment. The patient seeks medical advice because of vertigo and pains in the legs of three months' duration.

*Physical Examination.*—The patient has a leukoplakia of five years' duration. The eyes react to light and distance. An arcus senilis is present. The knee-jerks are exaggerated. The Babinski test and ankle-clonus are normal. No disturbance of sensation. Moderate degree of arteriosclerosis is present. The blood Wassermann is  $+$ <sup>4</sup>, as is likewise the spinal Wassermann. The Noguchi globulin test is positive. There are 20 cells to the cubic centimeter. The colloidal gold test is negative. The patient received twenty intravenous injections of arsphenamine. The Wassermann reaction after this treatment remained unchanged. The patient then received three modified Swift-Ellis intraspinal treatments, after which the blood Wassermann became reduced to a moderate positive. There was distinct improvement in the vertigo and pains in the legs. After three more intraspinal treatments the pains and vertigo disappeared. The blood Wassermann was moderately positive, the spinal Wassermann  $+$ <sup>4</sup>. He has now been without treatment for four months and still maintains his clinical improvement.

CASE X.—R. C. S., age thirty-six. Initial lesion fifteen years

ago. No internal treatment. Patient complains of staggering and shooting pains and loss of power in legs; great difficulty in walking. Duration of these symptoms three years.

Physical examination reveals absent knee-jerks and marked Romberg symptom. The blood Wassermann is  $+^4$ , as is also the spinal Wassermann. Cell count is normal. Noguchi globulin test negative. Collodial gold test normal.

The patient received twelve intravenous injections, with some improvement in his gait. The blood Wassermann remained  $+^4$ . The patient then received ten reinforced Swift-Ellis intraspinal treatments at intervals of two to three weeks. The blood and spinal Wassermanns still remained  $+^4$ .

The clinical improvement was, however, marked. The Argyll-Robertson pupil disappeared, the pupils reacting to light. (This is the only case in which we have observed the restoration of the pupillary reaction.) The pains in the legs disappeared and the gait became markedly improved. The extent of the improvement in walking may be appreciated when it is stated that whereas the patient leaned heavily on a cane when he first came under treatment, he later was able to completely dispense with it. The patient's station is improved, but the knee-jerks are still absent.

CASE XI.—J. T., age thirty-three. No history of infection. The patient complains of staggering and loss of power in the left arms. Duration of symptoms two years.

Examination disclosed absence of the knee-jerks and tendo achillis reaction. Argyll-Robertson pupil and Romberg symptoms present. There is distinct impairment of motor power in the left arm to such an extent that the patient cannot continue his work as a railway conductor. No loss of sensation. Moderate degree of arteriosclerosis. The blood Wassermann and the spinal Wassermann are  $+^4$ . The Noguchi globulin test is positive; the collodial gold test is negative; the cells were not counted.

The patient received eleven intravenous injections of arsphenamine, with but slight change in his physical condition. The blood Wassermann remained  $+^4$ . After the first modified Swift-Ellis spinal treatment the blood Wassermann was reduced

to a weak positive. The patient then received eight more intraspinal treatments at intervals of two to three weeks. After this treatment the blood Wassermann became negative, although the spinal Wassermann remained  $+$ <sup>4</sup>. There has been pronounced clinical improvement. The staggering is considerably improved and there has been recovery of the power in the arm to such an extent as to permit the patient to return to his former occupation. At the end of four months, without treatment, the improvement was maintained.

CASE XII.—D. D. G., age forty-seven. Initial lesion eighteen years ago. No secondaries observed. The patient took mercury by mouth for one year. During the past year the patient has had two intravenous injections of arsphenamine and a course of mercurial rubbings. The patient was brought to the hospital with paralysis of the lower extremities; complete inability to use the legs. The loss of power in the legs began a year ago, but has progressed rapidly during the past three months. The pupils are unequal, but react to light. The knee-jerks and Babinski are present. No loss of sensation. Diagnosis, myelitis syphilitica. The blood Wassermann is  $+$ <sup>4</sup>; the spinal Wassermann negative. The Noguchi globulin test and the colloidal gold test are likewise negative. Cells, 11 to the cubic centimeter.

The patient was given four modified Swift-Ellis treatments, after which the spinal Wassermann became a weak positive.

The clinical improvement has been remarkable, the patient is now able to walk without any support. The patient was given four more intraspinal and intravenous treatments, and left the hospital *with complete* restoration of power in the legs and able to walk perfectly.

The blood Wassermann was still  $+$  and the spinal Wassermann negative.

CASE XIII.—H. L., age thirty-one. Patient contracted syphilis in 1907; initial lesion and secondaries. Mercury was given by the mouth for one year. In 1913 the vision became blurred; later it improved without treatment, but subsequently he grew progressively worse and became totally blind in 1917.

*Diagnosis: Double optic atrophy.* Between March, 1916, and March, 1918, the patient received fourteen intravenous treatments of arsphenamine and four intraspinal treatments. The knee-jerks are absent. The blood and spinal Wassermann are both  $+$ <sup>4</sup>. The colloidal gold test and the Noguchi and Pandey globulin tests are negative. The cell count is 4 to the cubic centimeter. The patient received six further modified Swift-Ellis intraspinal treatments. Treatment was without avail and the patient is today totally blind.

CASE XIV.—I. B., age fifty-five. Patient complains of pains in both legs, as well as vertigo and difficulty in walking. There is also considerable vesical weakness. Duration of symptoms about thirty years (?).

Examination discloses Argyll-Robertson pupils and a positive Romberg. The knee-jerks are absent; no loss of sensation.

*Diagnosis: Tabes dorsalis.* The blood Wassermann is  $+$ <sup>4</sup> and the spinal Wassermann is  $+$ <sup>4</sup>. The Noguchi globulin test is negative; 8 cells to the cubic centimeter. The colloidal gold test is suggestive of a paretic curve. The patient received three intravenous injections of arsphenamine at weekly intervals, with but slight improvement. He then was given three modified Swift-Ellis treatments at intervals of two weeks. After this treatment the vertigo disappeared, the gait was markedly improved, and the patient increased greatly in strength. The blood and spinal Wassermans remained unaltered.

CASE XV.—B. H. W., age fifty-eight. Lues contracted about twenty-eight years ago. Treatment by mouth for a couple of years.

The patient came under the care of one of the writers eight years ago. At that time he had a severe osteoperiostitis of the left leg. He complained of general rheumatoid pains. He exhibited Argyll-Robertson pupils. Knee-jerks slightly exaggerated; station good. No bladder symptoms. Under intravenous injections of arsphenamine the osteoperiostitis and pains completely disappeared. The blood Wassermann was  $+$ <sup>4</sup>, and, despite over forty intravenous injections of arsphenamine and neo-arsphenamine, insoluble mercurial injections, and adminis-

tration of the iodids, the Wassermann has remained practically unchanged. In May, 1918, a spinal puncture was performed. The spinal Wassermann was negative; the Pandy and Noguchi tests positive. There were 3 cells to the cubic centimeter. The colloidal gold test exhibited a distinct luetic zone curve—0012221000. The patient was averse to receiving intraspinal injections, and, owing to his age, the matter was not pressed. His intellection is normal, his memory good, and there are no physical stigmata except the Argyll-Robertson pupil and a pronounced leukokeratosis of the tongue. The patient has a mild grade of myocarditis and has lost strength and weight during the past year, during which time he has had less treatment than in any period during the past eight years.

Persistent intravenous treatment combined with the use of mercurials appears to have prevented any marked advance in the nerve lesion during the past eight years. The blood Wassermann remains unchanged.

CASE XVI.—Dr. X, age fifty-six, presented himself two years ago complaining of tinnitus aurium and attacks simulating "petit mal," during which he would fall to the ground. On account of pronounced flushings of the face, several physicians had diagnosed vasomotor ataxia. The patient had no knowledge whatsoever of a luetic infection. His blood Wassermann was strongly positive. The patient was markedly neurasthenic and apprehensive. His pupils reacted well to light and the patellar reflexes were distinctly exaggerated.

The patient was given, during the course of a year and a half, over twenty intravenous injections of arsphenamine, mercury injections, and the iodids. Despite these treatments the patient's condition became worse. He developed paraphasia, employing inappropriate words in speaking. His speech became hesitating and incoherent, and his memory and his mind deteriorated to such an extent that it became necessary to remove him to a hospital. Here he was so irrational that he dipped his toothbrush into the urinal.

The spinal fluid was Wassermann negative; cells 8; negative globulin test; the colloidal gold test showed a distinct luetic zone

curve. Thirty-six hours after receiving a modified Swift-Ellis treatment his mind suddenly cleared up and his speech became normal. After two more intraspinal treatments he was dismissed from the hospital. Six months later, although still apprehensive and neurasthenic, his mind and speech were normal. The so-called "petit mal" attacks had ceased completely and the tinnitus aurium was distinctly improved. The blood Wassermann became negative.

#### RATIONALE OF THE TREATMENT

No conclusive deduction can be drawn as to the reason why the combined intravenous and intraspinal injections effect better results than the intravenous alone. The amount of arsphenamine in 15 c.c. of the patient's blood-serum is extremely minute. Even if  $\frac{1}{4}$  to  $\frac{1}{2}$  mg. is added *in vitro*, after the method of Ogilvie, the amount of the drug that would be thrown into the spinal canal would seem to be too small to accomplish the results achieved.

The explanation of the beneficial results may be that the blood-serum sets up a mild aseptic meningitis, and this process increases the permeability of the choroid plexus to the passage into the spinal fluid of the arsphenamine-charged blood plasma. Other physical conditions induced by the injection of blood-serum into the subdural space may likewise be operative. Dr. Francis X. Dercum, of this city, states that he has seen on many occasions improvement in the symptoms of nerve syphilis brought about by the injection into the spinal canal of plain blood-serum.

The histories above presented offer considerable material for commentary.

One of the most important questions in the controversy which has arisen as to the value of intraspinal medication in syphilis of the nervous system is, Will intraspinal treatment achieve that which cannot be accomplished by other means? For our own part, that query is answered by the results obtained in patients in whom intravenous medication was first tried with negative or mediocre results, and in whom combined intravenous and intraspinal treatment afterward brought about brilliant results.

Case XVI is a striking example of this. This patient had numerous intravenous injections of arsphenamine associated with the use of mercury and iodids administered in approved form. Not only was there no improvement, but the patient grew progressively worse and became incoherent and irrational. Thirty-six hours after the first combined intravenous and intraspinal treatment the patient's mind and speech suddenly cleared up, and have remained normal up to the present time—a period of seven months.

Case X, a case of tabes dorsalis, received twelve intravenous administrations of arsphenamine, with but slight improvement in his symptoms. Ten combined intravenous and intraspinal treatments effected an astounding change in the patient's ability to walk. After this the patient discarded the cane upon which he had relied for a long period.

Case XI exhibited but slight improvement in the muscular power of his arm after eleven intravenous treatments, whereas after nine combined intravenous and intraspinal injections he was restored to such efficiency as to enable him to resume his usual occupation.

#### INADEQUACY OF TREATMENT AND NERVE SYPHILIS

A striking feature of the above detailed histories is that practically all of the patients had totally inadequate treatment. In 25 per cent. of the patients the disease was unsuspected until nerve symptoms set in some years later. It is now realized that syphilis may occur without any local lesion recognizable clinically as a chancre. If it is ultimately proved that there is a special strain of spirochetes which have an affinity for nerve structure it may possibly be demonstrated that infection with such strains induces relatively little reaction in cutaneous and mucous tissues.

Another plausible explanation of unsuspected syphilis is that there may be a synchronous infection of gonorrhea and syphilis, the chancre being located in the urethra. Whatever the true explanation may be, the fact remains that we encounter many cases of late syphilis in patients in good social standing

who have no knowledge of a chancre or of any other antecedent manifestations.

During the secondary stage or period of vascular diffusion of the spirochetes these parasites are carried to the central nervous system, and either bring about inflammatory changes in a few months or years, resulting in the exudative forms of nerve syphilis, or in the course of years insidiously lead to inflammatory and degenerative types, such as tabes dorsalis and paresis.

#### THE BLOOD WASSERMANN AND THE SPINAL FLUID WASSERMANN

In this series of 16 cases of nerve syphilis the blood and the spinal Wassermann were studied in each case. The blood Wassermann was strongly positive in 15 cases. In one case of optic atrophy it was negative. The spinal fluid Wassermann was strongly positive in 11 cases; in one case of very early tabes with no symptoms save absent knee-jerks and inequality of the pupils, it was a weak to a moderate positive. In one case of meningomyelitis and one of transverse myelitis the spinal Wassermann was negative. It was likewise negative in a patient with long-standing syphilis exhibiting an Argyll-Robertson pupil and a resistant and strongly positive blood Wassermann. The spinal fluid showed increased protein and a distinct luetic zone curve with the colloidal gold test. In another case of pseudoparesis (probably a cerebral arteritis) the spinal Wassermann was negative.

In 6 cases of tabes dorsalis both the blood and spinal Wassermanns were strongly positive. Cases in which nerve syphilis exists with a negative blood Wassermann are not, however, rare. In a patient under our care at the present time, who exhibited evidences of a profound neurasthenia, there was no history of a syphilitic infection and the blood was Wassermann negative. The spinal fluid, however, was strongly positive. A prominent neurologist who examined this patient believes him to be suffering from incipient paresis.

Another patient, thirty-five years of age, with treated syphilis of two years' standing, presented himself with neurasthenic

symptoms and a negative blood Wassermann. A spinal puncture revealed a strongly positive Wassermann. The patient delayed treatment and a few weeks later developed a hemiplegia.

Cases such as this indicate the imperative necessity of diagnostic spinal puncture in all cases of syphilis. Surely no case of syphilis should be released from observation without an examination of the spinal fluid.

#### SIGNIFICANCE OF LABORATORY FINDINGS

The existence of a strongly positive blood Wassermann indicates the presence of living spirochetes in the body. A strongly positive spinal Wassermann is evidence of the involvement of the central nervous system.

The evidential value of a negative blood Wassermann or a negative spinal Wassermann is much less conclusive than that of a positive reaction. We know definitely that the blood may be negative in the presence of indubitable syphilitic lesions; the same statement may be made with reference to the spinal Wassermann test.

A marked increase of the cells in the spinal fluid indicates exudative or inflammatory changes usually involving the meninges; the greater the number of cells, the more pronounced the inflammatory activity.

Lange's colloidal gold test is a valuable addition to our laboratory technic. It is of value in indicating a pathologic state of the spinal fluid, even when the cells, protein and Wassermann, are negative. It is also of differential diagnostic and prognostic value. It is of special usefulness in differentiating tabes and cerebrospinal syphilis from paresis. Its findings are not absolute, however, as a characteristic paretic curve may occasionally occur in cases of cerebrospinal syphilis. Wisdom and experience both indicate that clinical and laboratory findings should be weighed side by side and deductions drawn from a careful study of all of the available evidence. Such a course will lessen errors of diagnosis, prognosis, and treatment.

### THE EFFECT OF TREATMENT UPON THE CLINICAL AND LABORATORY FINDINGS

While it is true that a certain relationship exists between the extent of the clinical manifestations and the intensity of the pathologic findings in the spinal fluid, yet there are many exceptions to this generalization. Both the clinical and the laboratory findings may be favorably influenced by treatment, but there is often an absence of parallelism in the improvement. The clinical symptoms are usually influenced to a greater extent than the pathologic findings.

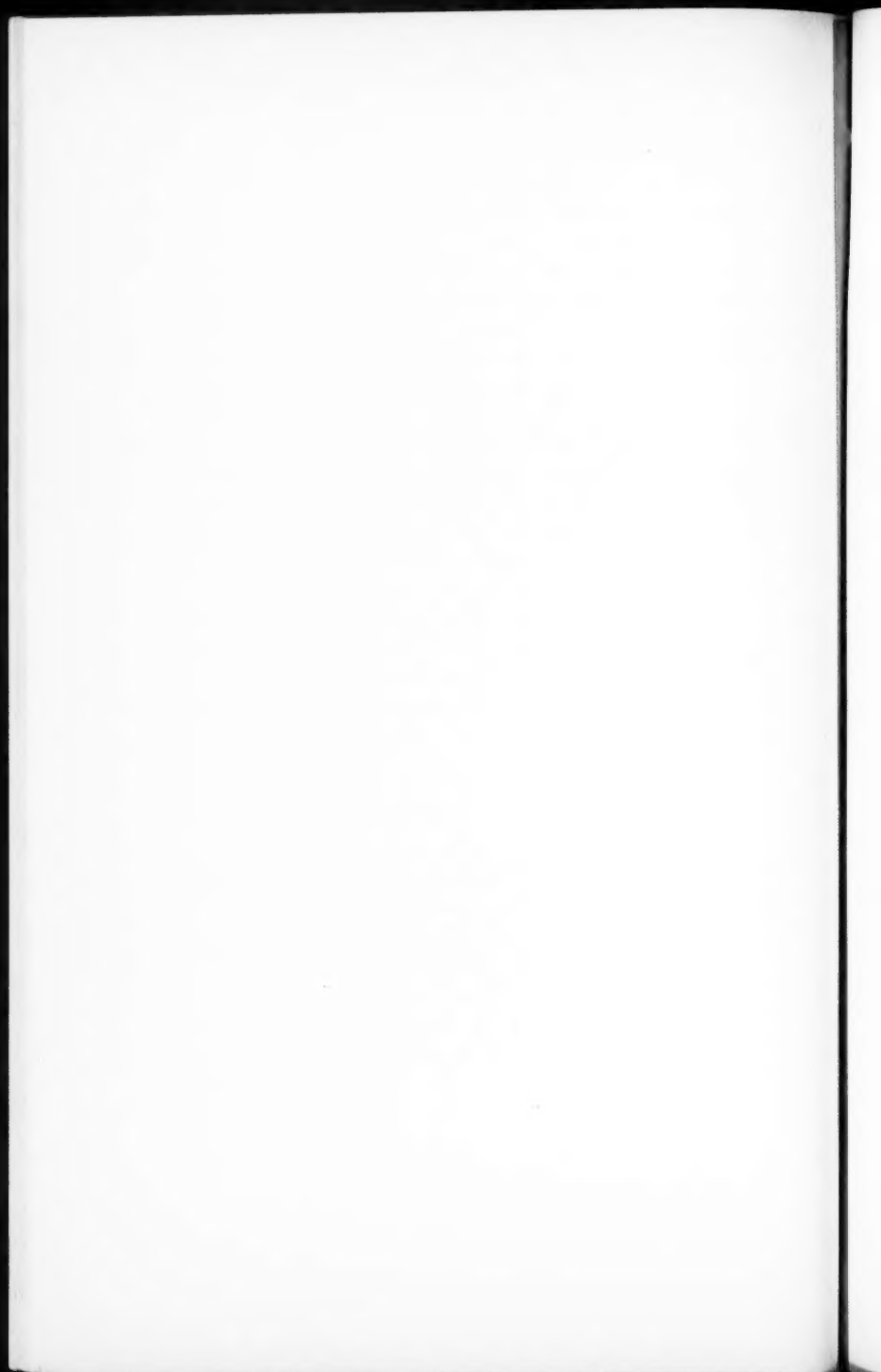
Sensory and motor disturbances are apt to yield first to treatment. Sexual power, when impaired, is commonly strengthened; the gait is often astonishingly improved. The mobility of the pupil may be bettered, and in rare cases an Argyll-Robertson pupil may be made to disappear. It is extremely difficult to influence a lost patellar reflex. These improvements may take place while no change occurs in the laboratory findings of the spinal fluid. In other cases a pronounced and progressive improvement in the spinal findings may take place under systematic treatment. The cellular count is commonly reduced after a few treatments. Influence upon the Wassermann and gold test is more slowly effected.

We have had several cases in which intraspinal treatment produced a negative blood test after intravenous treatments alone had failed to achieve this result.

We have also observed in several instances what appeared to be a provocative spinal Wassermann, the reaction being negative before treatment and positive afterward. The goal to be aimed at should be the disappearance of clinical symptoms, and the extinction of the pathologic findings in the blood and spinal fluid. In some cases this consummation, devoutly to be wished, cannot be achieved. Patients are often grievously disappointed because the blood or the spinal Wassermann remains positive despite repeated courses of treatment. Under such circumstances they should be informed that each added course of treatment increases the insurance against future disability.

In conclusion, a word must be said about the limitations of

intraspinal therapy. One can never hope to restore degenerated nerve tissue. In advanced cases of paresis or tabes intraspinal treatment may, in some instances, aggravate, rather than improve, the condition of the patient. Paresis cannot be cured unless treatment is begun at an extremely early stage. The most favorable time for treatment is before clinical evidence of paresis develops. A routine examination of the spinal fluid in all cases of syphilis would enable physicians to detect the pre-paretic stage by the spinal fluid findings. Systematic treatment at this time would doubtless prevent this unfortunate eventuality. The same statement is to a greater extent true of other forms of nerve syphilis which are more amenable to treatment. We must have numerous well-equipped institutions and well-trained men to carry out the arduous work which such systematic examinations and treatments would entail.



## CLINIC OF DR. ELMER H. FUNK

JEFFERSON HOSPITAL

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### CHYLOTHORAX

**Patient No. 1.—Chylothorax. Introduction. True and Spurious Forms. Frequency. History and Clinical Notes of Patient. Discussion. Anatomy of Thoracic Duct. Etiology of Chylothorax. Clinical Manifestations and Diagnosis. Prognosis and Treatment.**

INSTANCES of effusion in the serous cavities having a milky appearance are decidedly uncommon. Such collections are met with most often in the peritoneum, less frequently in the pleura, and only very rarely in the pericardium. Milky effusions in the pleural cavity are so rare that many clinicians of experience have never seen a case. Bartolet in 1633 was the first to describe an effusion of this type. In discussing milky pleural effusions it is well to keep in mind that they are of two principal varieties: (1) the true chylous, in which, as a result of a lesion of the thoracic duct, chyle finds its way into the pleural cavity, and fat is present in the fluid, and (2) the pseudochylous, in which the milky appearance is not due to fat, but to a lecithin globulin complex held in suspension by the inorganic salts present (Wallis and Schölberg). Some writers have added a third variety—the chyliform—in which the milky appearance of the effusion is due to the fat present, resulting from fatty degeneration of cellular elements. Henry states that it is questionable whether the milky appearance can be entirely due to this cause. Only the first variety represents true chylothorax and is the form we are discussing today in the presentation of our patient.

Lewin in 1916 was able to find only 51 cases of chylothorax in the literature since Bartolet's time. F. T. Lord is of the opinion that they are probably more common than the number of

reported cases would indicate, since chylous may be readily confused with purulent fluid unless carefully examined. Since 1916 I have been able to find two reports in the literature (Pisek, 1917; Brekke, 1917), making 54 cases to the present time. The following is the history and clinical notes of this patient:

T. S. B., male; colored; aged forty-two years; physician; admitted to the Jefferson Hospital August 28, 1918.

*Chief Complaint.*—Shortness of breath upon slight exertion.

*Family History.*—Father died at eighty-four years of apoplexy. Mother, six brothers, and two sisters are living and well. One brother died at twenty-four years of typhoid fever.

*Personal History.*—Measles, mumps, chicken-pox, and whooping-cough in childhood; typhoid fever complicated by phlebitis of left leg at twenty-two years; rheumatic fever with prompt convalescence at twenty-six years; diphtheria at thirty-five years.

*Present Illness.*—One month prior to admission he noticed an increasing shortness of breath upon exertion, associated at times with slight vertigo. He was also distressed with palpitation upon exertion; otherwise he had no complaint—no pain, cough, night-sweats, or fever were evident. About ten days after the onset he took to his bed because of weakness and dyspnea, and was examined by a fellow practitioner, who diagnosed the presence of fluid in the left chest. He was aspirated and about 4 quarts (!) of milky fluid withdrawn. The aspiration was followed by a prompt relief from symptoms, which lasted for three or four days, and then returned as the fluid reaccumulated. One week later he was aspirated again and 3 quarts (!) of milky fluid removed. This aspiration, like the first, was followed by relief from the shortness of breath and palpitation for a few days, but they returned again shortly before admission to the hospital.

*Physical Examination.*—Patient is a well-developed, well-nourished adult male. The eyes, ears, nose, mouth, and throat are negative. Examination of the neck reveals no glandular enlargement. The chest is well developed and shows some bulging on the left side below. The expansion on this side is restricted, especially in the lower portion; over the entire left base are flatness, diminished vocal fremitus and resonance, and dis-

tant breath sounds. No râles are audible anywhere in the chest. The heart is moderately displaced toward the right. The heart sounds are fairly good; no murmurs are audible. The abdominal examination is negative. The extremities are negative. The urine is negative. The sputum shows no tubercle bacilli.

*Progress.*—Five days after admission the patient was quite dyspneic, and it was thought advisable to aspirate—3000 c.c. of milky fluid were removed from the left chest. Following the aspiration the patient was considerably relieved. The aspirated fluid was examined, with the following result: Specific gravity 1023, total nitrogen 0.70 per cent., protein 4.38 per cent., fat 1.8 per cent., sugar 0.11 per cent. There was considerable fibrin present. This was removed before the determination for nitrogen and protein was made.

*Discussion.*—Before discussing those conditions which may produce chylothorax, it may be well to refresh our memory briefly with regard to the anatomy of the lymphatic vessels as they pass through the thorax. The lymph coming from all parts of the body is collected into two tubes—the right lymphatic duct and the thoracic duct. The *right lymphatic duct* is smaller and collects the lymph from the right side of the head and neck, right arm, right side of chest, and the upper convex surface of the liver. The various lymphatics unite to form a duct 1 or 2 cm. long which empties into the venous system at the junction of the right internal jugular and subclavian veins. Of greater importance, because it conveys chyle in addition to a great mass of lymph into the blood, is the left lymphatic duct, or *thoracic duct*. It originates in the abdomen at the receptaculum chyli at the level of the second lumbar vertebra; enters the thorax through the aortic opening of the diaphragm to the right of the aorta, and traverses the posterior mediastinum between the aorta and vena azygos major. As it ascends it lies on the bodies of the seven lower thoracic vertebræ with the pericardium, esophagus, and the arch of the aorta in front. At the level of the fifth thoracic vertebra it inclines toward the left side, ascends behind the arch of the aorta on the left of the esophagus, and behind the first portion of the subclavian artery. At the level of the

seventh cervical vertebra it turns outward and then downward over the left pleura, subclavian artery, and scalenus anticus, and empties into the venous system at the junction of the left internal jugular and subclavian veins. At its commencement there is a dilatation called the receptaculum chyli, and in its course it is flexuous and constricted at intervals, giving to it a varicose appearance. Not infrequently it divides in the middle of its course into branches which reunite, forming a plexiform interlacement. It has valves throughout its course which are more numerous and more competent in the upper portions, and a very efficient pair is present at its termination to prevent the passage of venous blood into the duct. In some instances there are several terminations of the duct, and Wendel believes that it may anastomose with the right lymphatic duct and azygos veins, explaining thereby the lack of ill effects when it is ligated following injury in operations in the neck.

The *causes* of chylothorax are either (1) rupture of the thoracic duct or its radicles or (2) increased pressure within the duct leading to a backward flow of lymph along the pulmonary and pleural lymphatics. West states that in the majority of cases rupture is due to injury, but in a few to disease, and it is probable that the duct may be ruptured without much external sign of serious damage. West further calls attention to the fact that the thoracic duct may not be at fault, the lesion occurring in one of the larger lymphatics elsewhere in the thorax. Among the 47 cases collected by Baldwin in 1908 the causes were as follows:

	Cases.
Chest injury.....	16
Pressure upon duct by new growths or tuberculous glands.....	9
Secondary growths in duct.....	9
Thrombosis of left subclavian vein.....	4
Proliferating lymphangitis.....	2
Aneurysmal dilatation of duct.....	2
Thrombosis of duct.....	1
During operation for removal of carcinomatous glands of neck.....	1
Obstruction of duct from inflammatory thickenings in the mesenteric.....	1
Mitral disease.....	1
Filaria.....	1
Total.....	47

The *clinical manifestations* of chylothorax are those of simple serous effusion, and the diagnosis is made by the exploring needle. Its nature is rarely suspected until the milky fluid is obtained, and then it may be mistaken for a purulent collection. A careful microscopic and chemical examination of the fluid will decide. There are seldom any subjective symptoms in the non-traumatic cases other than gradually increasing dyspnea and weakness. In our patient these were the only symptoms. Fever is usually absent, as in our case. The majority of the patients are adult males, although the condition may occur in infancy. Jennings reported before the American Pediatric Society in 1907 the case of a child of nine months in whom the aspirated fluid from the chest contained 3 to 4 per cent. of fat. The majority of cases of chylothorax occur on the right side (Edwards 60 per cent.), and in approximately one-third of the cases there is an accompanying chylous ascites. In our patient the accumulation was left sided and there was no demonstrable ascites. In the patient recorded by Rokitsky the chylothorax was bilateral and associated with chylous ascites. In de Mussy's patient the condition existed on the left side for fifteen years and was followed by recovery after three tapplings. In Erb's patient 30 liters of chylous fluid were removed in eight tapplings. Massing's patient presented bilateral chylothorax, chyloperitoneum, and chylopericardium.

The differentiation of true chylothorax from pseudochylous hydrothorax may at times present difficulties. The fluids may have a similar milky appearance. A milky fluid is more likely to be a true chylous rather than a pseudochylous fluid.

A true chylous fluid tends to accumulate rapidly, contains microscopically fine fat globules which stain readily with osmic acid and Sudan III; there are very few cellular elements. The specific gravity generally exceeds 1012, and although the total solids show considerable variations, a value below 4 per cent. is rarely found. The fat content is generally high, varying from  $\frac{1}{2}$  to 4 per cent. The percentage of fat present in this case was  $\frac{8}{10}$  per cent. Lecithin occurs only in traces.

In pseudochylous effusions the fluid tends to accumulate less

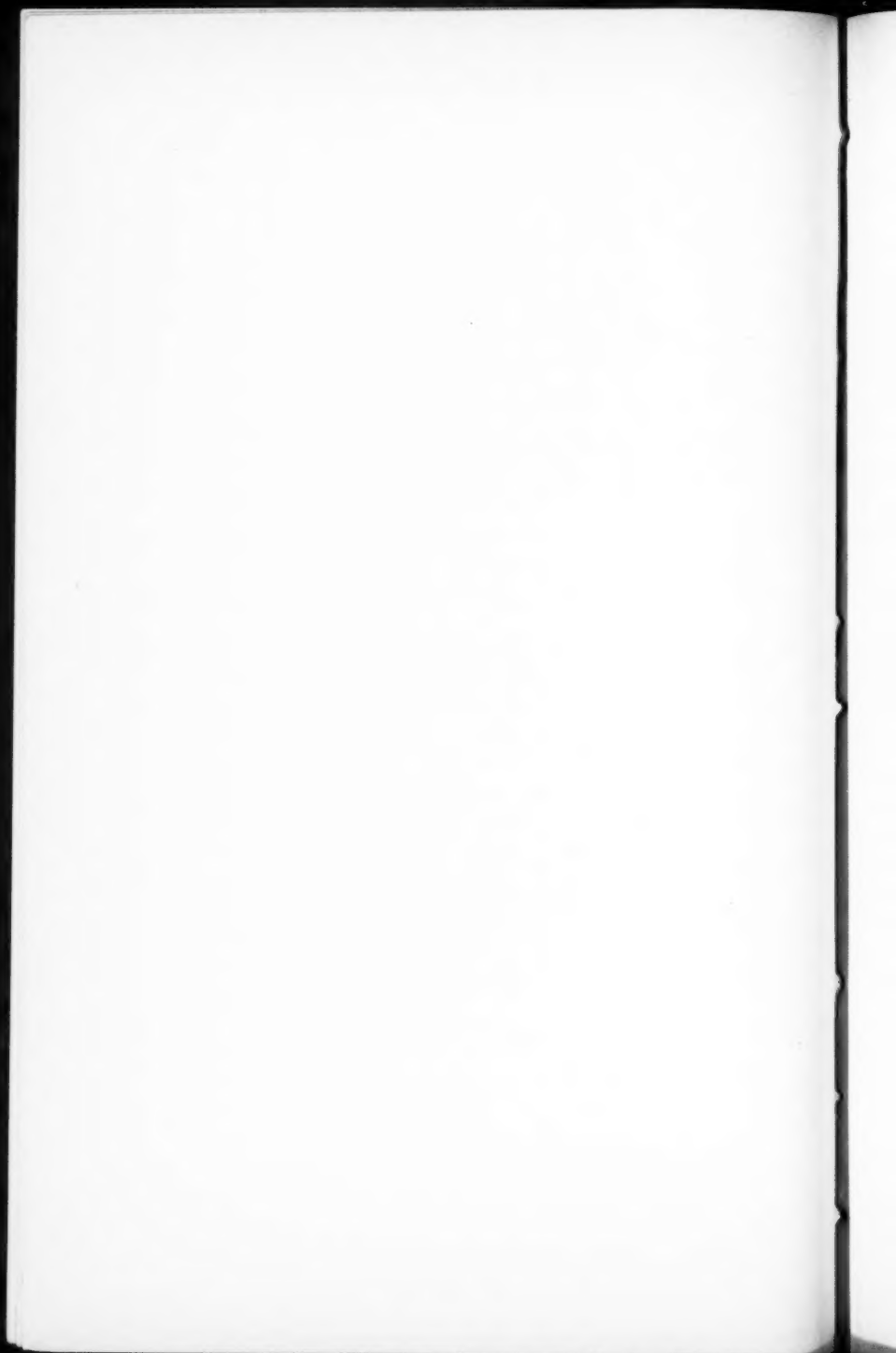
rapidly, contains microscopically numerous fine highly refractile granules which do not give the reactions for fat. The cellular elements may be numerous and often contain fat. The specific gravity is usually less than 1012, and the total solids rarely exceed 2 per cent. The fat content is low and in many instances only a trace can be found. Lecithin is present in combination with globulin. This lecithin-globulin complex is the cause of the milky appearance of the fluid. Pseudochylous fluids resist putrefaction. This characteristic has been attributed to the lecithin. To those of you who wish to look into the matter of the differentiation of chylous and pseudochylous fluids, in more detail than I have given, I would recommend the very excellent articles by Wallis and Schölberg which appeared in the *Quarterly Journal of Medicine* for 1910 and 1911.

**Prognosis and Treatment.**—The perforation of the duct following injury or disease may close spontaneously if the opening is small. Radical treatment by operation upon the duct is not feasible at the present time in intrathoracic cases; in those instances in which the injury results from operation in the neck the duct may be ligated. Accidental injury of the duct in this way has occurred in the experience of many surgeons, and practically always without unfavorable results when ligated or packed. The inference has been that there are several terminations of the duct, or, as Wendel has pointed out, it anastomoses with the right lymphatic duct or with the azygos veins. The accumulation of chylous fluid in the thorax requires tapping if pressure symptoms are present, or if after a moderate wait the effusion shows no evidence of being absorbed. The fluid should not be entirely removed at one time or the tapping performed too frequently, as a certain amount of pressure may be necessary to prevent the escape of more fluid from the duct and to favor repair. Many of the reported tappings show large removals, frequently resorted to, and with an unfavorable termination. In fact, at the present time the treatment of chylothorax is most unsatisfactory. Treatment of the underlying condition is indicated, and if this is tuberculosis, the usual rest in bed with an abundance of fresh air and good nourishing food is essential.

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<sup>1</sup> Articles by Lewin and Wallis and Schölberg contain excellent bibliographies, by consulting which the literature is rendered readily available.



## AORTIC ANEURYSM WITH ESOPHAGEAL RUPTURE

Patient No. 2.—Aortic Aneurysm. History and Clinical Notes of Patient. Death by Rupture into the Esophagus. Autopsy. Discussion of Esophageal Symptoms of Aneurysm and the Relative Frequency of Esophageal Rupture. Early Diagnosis of Aneurysm.

CLINICAL history and notes of H. C., male, white, aged fifty-two years; admitted to the Jefferson Hospital August 26, 1915:

*Chief Complaint.*—Cough of one year's duration and pain in the back between shoulders.

*Family History.*—Negative.

*Personal History.*—Chicken-pox at eleven years. Gonorrhea seven times, first when fifteen years old. He had a chancre in 1900, that is, fifteen years ago, followed by a rash over his entire body. A diagnosis of syphilis was made, but he received no systematic treatment. He states that he has always been in good health until the present trouble.

*Present Illness.*—One year ago he caught cold while working in the early morning on a farm, and the resulting cough has persisted until the present time. The cough is paroxysmal in character and is worse on some days and better on others. It is associated with a slight expectoration of a yellowish, mucoid material. The pain in the back is high up between the shoulders and is quite severe at times. It has been more or less constantly present. About six months prior to admission he was given a three weeks' course of potassium iodid, following which he felt considerably better.

*Physical Examination.*—Patient is a well-nourished, well-developed adult male. No cyanosis. No dyspnea. The pupils are equal and react to light and accommodation. The ears and nose are negative. The tongue is coated with a whitish fur. The teeth are in bad condition. In the neck a distinct tracheal tug can be obtained. The chest is well developed, although ex-

pansion is limited on both sides. The percussion note over the lungs is normal, the breath sounds quiet, and scattered piping râles are audible over the entire pulmonary area. The heart examination reveals the apex normally placed, no shock or thrill, no expansile pulsation with one hand on the front of the chest and the other hand on the back. The heart sounds are clear and distinct and without murmurs. The liver and spleen are not enlarged. There is a right incomplete inguinal hernia. The patella reflexes are absent.

*Laboratory Tests.*—The urine is of low specific gravity, contains a faint trace of albumin and a few granular casts. The blood shows Hb. 72 per cent., R. B. C. 5,230,000, W. B. C. 9200; differential count—large mononuclears 3 per cent., small mononuclears 36 per cent., polynuclears 60 per cent., eosinophils 1 per cent. The sputum shows no tubercle bacilli. The Wassermann test is positive, 4+. The x-ray examination shows a small aneurysm involving principally the first portion of the descending arch of the aorta.

*Progress.*—While in the hospital he remained for the greater part of the time in bed or in his chair. He complained a great deal of the pain in his back and of the distressing cough, which since admission had become quite productive of a yellowish, mucoid sputum. Three days before death he spat blood which seemed to come from the respiratory passages, because it occurred during the act of coughing. He had never vomited. On the morning of his death he seemed in his usual condition, although he stated he felt disinclined to eat. He was sitting on the edge of his bed when he called for the nurse, stating that he felt faint. He was urged to lie down, and in about fifteen minutes became quite pale. By the time the resident physician reached his bedside, in about ten or fifteen minutes more, he was dead, presumably with an internal hemorrhage. Some little blood exuded from his mouth while he was dying. There were no respiratory symptoms and we were led to a provisional diagnosis of rupture into the esophagus.

*Autopsy.*—The body was turned over to the State Anatomical Board because of lack of claim, and the Board sent it to our

Anatomical Institute, where cross-sections of the cadaver were made. These sections revealed the aneurysm with its esophageal relationship and the rupture into this structure. The esophagus, stomach, and intestines were filled with blood. The accompanying illustrations will show better than words the autopsy findings.

**Discussion.**—Symptoms referable to the esophagus are not of frequent occurrence in patients with thoracic aneurysm. In a few instances the early extension of the sac backward may give rise to dysphagia before other signs of the aneurysm are demonstrable. Cases are on record in which esophageal instruments have been passed for diagnosis, or for treatment, and the thin wall between the esophagus and aneurysm penetrated, resulting in immediate death from hemorrhage. Fortunately, such occurrences nowadays are rare, the fluroscope and the x-ray plate rendering the early diagnosis of aneurysm a relatively easy matter. A dysphagia present early in the development of an aneurysm may not be due to pressure, but to spasm due to pressure on the recurrent laryngeal nerve. A rather large-sized aneurysm may not give rise to any interference with the passage of food, or, just the opposite, may cause the greatest difficulty and, if ulceration is present, agonizing pain. In other patients the proximity to and involvement of the esophagus may not be suspected until a hemorrhage occurs. The perforation may not be fatal. If the opening is small and a clot forms, the perforation may close. If the opening is large, fatal bleeding may occur into the esophagus and externally, or the blood may pass into the stomach without appearing externally. Such a condition may be discovered only at autopsy.

In our patient aortic aneurysm was suspected because of the persisting pain, the harsh metallic cough existing for a year without demonstrable lung lesion in a man of fifty-two years with a history of syphilis, and in whom a definite tracheal tug was obtainable. This diagnosis was made in spite of the fact that no paramanubrial dulness or other local sign of aneurysm could be found on physical examination. The x-ray verified the diagnosis. The autopsy revealed its anatomic relation and showed

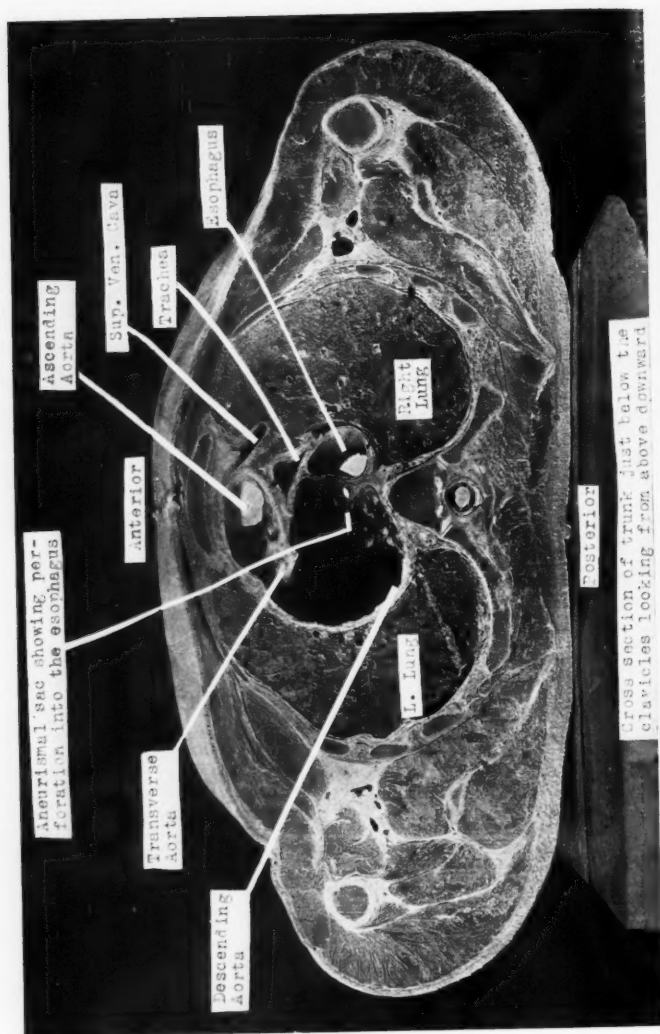


Fig. 142.

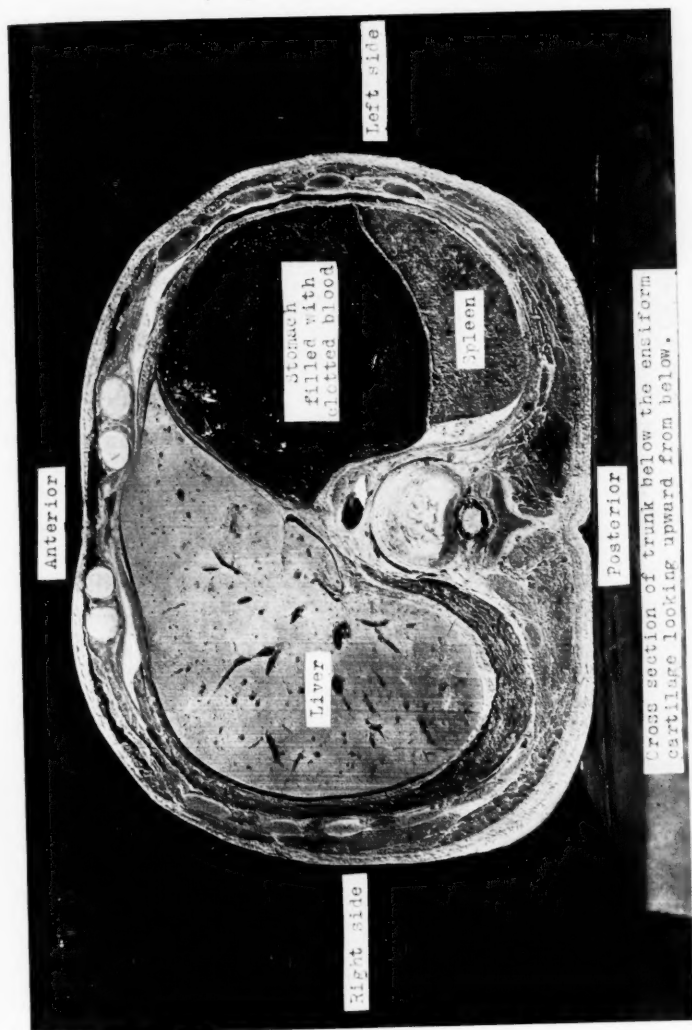


Fig. 142.

Fig. 143.

the cause of death to be due to perforation into the esophagus with profuse bleeding into the stomach and intestines.

It is not my purpose to discuss with you the diagnosis of aortic aneurysm, except to state that unless you keep its possibility constantly in mind you will miss it many times. It has been said that nothing is more conducive to clinical humility than aortic aneurysm, certainly the autopsy table reveals its great frequency, and in recent years this has been emphasized again by the x-ray studies of the chest. In every adult complaining of persisting pain in the chest, either anteriorly or posteriorly, particularly men over thirty years with a past history of syphilis, the possibility of aortic aneurysm must be kept in mind. Autopsy figures reveal the frequency of the disease which clinically is not demonstrable by physical signs, at least by but few physical signs, such as slight increase in the manubrial dulness, etc. The "latent" types, those with a few symptoms or signs, are not an infrequent cause of sudden death. If we expect to find the classical text-book symptoms and signs we will miss most of the cases of aneurysm. It is the partially developed types, those with a few symptoms and no signs, or a few signs and no symptoms, which form the great group of cases. The "aneurysm of symptoms," rather than the "aneurysm of signs," is the type we overlook.

I want to call your attention to a sign which was present in our patient and which ought to be sought for in all patients in whom a deep-seated aneurysm is suspected. The sign was described by Surgeon-Major Oliver, and is spoken of as tracheal tugging. It is elicited, in the words of Dr. Oliver, as follows: "Place the patient in the erect position, and direct him to close his mouth and elevate his chin to almost the full extent; then grasp the cricoid cartilage between the finger and thumb, and use steady and gentle upward pressure on it, when, if dilatation or aneurysm exists, the pulsation of the aorta will be distinctly felt transmitted through the trachea to the hand." The elevation of the chin is not necessary and it may sometimes be elicited more easily with the chin down. Tracheal tugging is due to the transmission of the pulsations of the aortic aneurysm as a result

of compression of the left bronchus, or as a result of adhesions between the sac and the trachea or bronchi. It is not pathognomonic, inasmuch as it has been found in simple dilatation of the aorta and in mediastinal growths.

The cause of death in patients with thoracic aneurysm is due to rupture in at least one-half of the cases. The structures involved in the rupture were studied by Lemann, who found among 592 collected cases the following order of frequency: pericardium alone, 148; left bronchus, pleura, or lung, 160; right bronchus, pleura, or lung, 62; esophagus, 50; trachea, 48; superior vena cava, 31; pulmonary artery, 18; other structures, 40. The relative frequency of rupture into the esophagus, as in our patient, is variously stated by different writers, thus among almost 600 cases collected or reported by Borowsky, Cush, Huchard, Charcov, Oswald, Browne, Kelynod, Hall, and Biggs, it occurred 50 times, varying with the individual writer from 1 in 7, as reported by Hall, to 1 in 24, as reported by Biggs.

Finally, before we pass on to the discussion of our next patient, I wish to emphasize to you again the relationship which syphilis bears to cardiovascular disease. As in our patient, so in all patients, syphilis as a predisposing factor to aneurysm holds first place. And not only is this important in the etiology of aneurysm but also in the etiology of cardiovascular diseases in general. In every old case of syphilis the aorta when examined microscopically shows characteristic syphilitic infiltration of the media and adventitia—changes which may not be visible to the naked eye. In the early stages of syphilis, as Warthin has demonstrated, slight or no changes are evident microscopically, and yet the characteristic lesions with spirochetes are demonstrable microscopically. The microscopic picture is that of "small infiltrations of lymphocytes and plasma cells along the vasa vasorum of the media and adventitia, usually most marked around the vessels of the latter, and diminishing in degree as the vessel passes up into the media. Perivascular proliferation, fibrosis, and obliteration of the small vessels then follow. The resulting disturbances of nutrition of the vessel wall are first seen in the intima and the inner portion of the media, in the form

of fatty degeneration, atrophy, and necrosis of the cells of this portion of the vessel, with weakening and thinning of the wall, followed later by fibrosis and hyaline change. The involvement of the media progresses steadily outward, and because of the greater involvement of the media locally there results local thinning of the vessel wall and microscopic ruptures of the elastic fibers. Such changes predispose to the development of aneurysms" (Warthin).

## TUBERCULOSIS AND PREGNANCY<sup>1</sup>

Brief Records of 5 Patients. Discussion. The Influence of Pregnancy on Tuberculosis. The Influence of Tuberculosis on Pregnancy. Conclusions as to the Treatment of Pregnancy in a Tuberculous Patient. Care of the Child.

THE attitude which physicians should take in advising a tuberculous patient who has become pregnant depends so much upon the consideration of the individual patient that it is difficult to formulate any definite rules of action. Our conception of the relationship of these two conditions and our plan of action are illustrated by the case records of the following patients:

CASE I (G-4028).—A. W., aged twenty-seven years, admitted to the Jefferson Hospital Department for Diseases of the Chest January 10, 1917.

*Family History.*—Negative.

*Personal History.*—Married five and a half years. Two children living and well.

*Present Illness.*—In April, 1916 (about nine months ago), when patient was four months pregnant, she noticed that she was losing in weight and strength and had a slight cough, for which she consulted a physician, who said she had bronchitis. After the baby was born (five months ago) she became rapidly worse, with aggravation of cough and expectoration, with the addition of chilliness, fever, and night-sweats.

*Physical examination* upon admission revealed advanced bilateral tuberculosis of the lungs, with cavity formation. Temperature 101° to 103° F. in the afternoons, with chilliness and sweats. The sputum showed many tubercle bacilli.

<sup>1</sup> A reference to the more important articles in the literature may be found in the author's article on the Relationship of Tuberculosis and Pregnancy which appeared in the *Therapeutic Gazette* March 15, 1915. An excellent reference with regard to the treatment of these patients is that by Norris and Landis, Fourteenth Report of the Henry Phipps Institute, 1918.

**Progress and Comment.**—The patient became progressively worse, and died three months after admission, or eight months after delivery. The history is clearly that of onset of symptoms of tuberculosis during pregnancy, with rapid decline after delivery.

**CASE II (G-2029).**—F. T., aged twenty years; admitted to the Jefferson Hospital Department for Diseases of the Chest September 23, 1916.

*Family and previous personal histories* contain nothing noteworthy. Married when seventeen years old; has one child seventeen months old.

*Present Illness.*—Eighteen months ago, during the latter months of pregnancy, she began to cough, expectorate, and lose weight. Shortly after delivery she recovered to some extent, the cough lessened, and she gained in weight. She was definitely improving with regard to the pulmonary lesion, and was advised against conception, at least until such future time as it could be done with reasonable safety. In spite of this advice she became pregnant, and now at five months has a return of the cough, expectoration, and fever. She has lost about 30 pounds in weight and four days ago she had a small pulmonary hemorrhage.

*Physical examination* revealed moderately advanced and active bilateral tuberculosis of the lungs. The sputum contains many tubercle bacilli.

**Progress and Comment.**—The patient was placed under observation for one week. Because of the acuteness and advanced stage of the tuberculous lesion, and the advanced stage of the pregnancy, it was deemed inadvisable to interfere. The patient died six weeks after admission. A study of the patient's history emphasizes the need of warning against conception in one who has had a recent pulmonary lesion, as this woman had, and the dire results with which failure to abide by this advice is fraught. The first pregnancy activated, in all probability, an old latent tuberculous lesion. Following delivery there was definite evidence that the disease was tending to subside, when the second pregnancy intervened, causing an exacerbation of the tuberculous disease, resulting in death during pregnancy.

**CASE III (G-10844).**—D. B., aged twenty-four years; ad-

mitted to the Jefferson Hospital Department for Diseases of the Chest September 25, 1917.

*Family History.*—One brother died of tuberculosis.

*Personal History.*—Married three years ago. One child living and well. At the present time five months pregnant.

<b>JEFFERSON HOSPITAL</b>		Highest Weight..... Average Weight..... Lowest Weight.....
DEPARTMENT OF DISEASES OF THE CHEST PHILADELPHIA, PA.		
<b>WEIGHT CHART</b>		
Name <u>Dora Berkman</u>	Admitted <u>9-27-1917</u>	

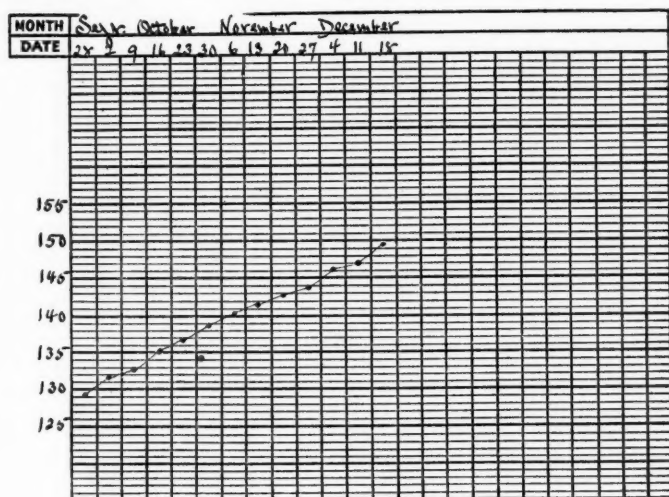


Fig. 144.

*Present Illness.*—Two months ago began to cough, which has continued to the present time, with expectoration and loss of 13 pounds in weight.

*Physical examination* revealed bilateral pulmonary tuberculosis, with most marked involvement on the right side, where below the right clavicle tubular breathing, whispering pectoriloquy, and scattered crackling râles suggested considerable involve-

ment and activity. The sputum was positive for tubercle bacilli. The patient, however, stated that she had a good appetite and felt fairly well, except for the cough. She had no fever.

**Progress and Comment.**—We decided, in view of the fact that the patient was fairly well along in her pregnancy, and because of the chronic nature of the tuberculous lesion, to place her under observation and be guided by the subsequent course as to whether we would or would not terminate pregnancy. We soon found that in spite of what seemed to be alarming physical signs, she gained progressively in weight (Fig. 144) and remained absolutely afebrile, and although the cough persisted, she improved so much that we were able to take her to term in better condition than when she came under observation. She was delivered of a healthy baby, and at the present writing, about one year afterward, she is, to all intent and purposes, an arrested case, and with a regulated life is useful in the care of her home.

**CASE IV (G-2065).**—R. B., aged eighteen years, admitted to the Jefferson Hospital Department for Diseases of the Chest November 8, 1916.

*Family History.*—Negative.

*Personal History.*—Married nine months ago. At the present time not quite four months pregnant.

*Present Illness.*—Two months ago began to cough and expectorate and lose weight.

*Physical examination* revealed early active and bilateral pulmonary tuberculosis. Sputum showed many tubercle bacilli.

**Progress and Comment.**—The termination of pregnancy was advised and done under the skilful attention of an experienced obstetrician. One month following the delivery the cough and expectoration lessened, and at the present time, two years later, she is in excellent condition.

**CASE V (G-10896).**—F. C., aged nineteen years; admitted to the Jefferson Hospital Department for Diseases of the Chest November 29, 1917.

*Family History.*—Negative.

*Personal History.*—Married four years; one child living and well. At the present time seven months pregnant.

*Present Illness.*—Two months ago began to cough, expectorate, and lose weight, and had horseness at times. Slight fever.

*Physical examination* revealed limited bilateral pulmonary tuberculosis, with a few crackling râles. Sputum positive for tubercle bacilli.

**Progress and Comment.**—Under observation and with rest and hygienic dietetic care the lesion in the lung seemed to be progressing. After consultation, the obstetrician decided to induce labor prematurely. This was done, and a living child secured. The activity of the disease in the mother promptly subsided, and at the present writing, one year afterward, both the mother and child are doing very well.

**Discussion.**—The association of tuberculosis and pregnancy is not uncommon; in fact, among every group of married tuberculous women, the history is frequently obtained that the tuberculosis manifested itself for the first time during or following a recent pregnancy. Among 200 married women with pulmonary tuberculosis admitted to our wards during the past five years approximately 30 per cent. gave this history. It is entirely probable that many of the patients included in this percentage were actively tuberculous before conception, and the occurrence of pregnancy intensified the process to such an extent as to call attention to the disease which had hitherto been unrecognized. It is fairly generally conceded that tuberculosis is a childhood infection and that its clinical manifestation in adult life, oftentimes many years afterward, is a result of some factor or factors which reduce the general or local resistance.

According to postmortem evidence and the tuberculin test it has been shown that practically all those who have reached fourteen years of age have been infected. The occurrence of tuberculous manifestations in later years results from a local or general lowering of the tissue resistance. In view of the great frequency of early tuberculous infection, and the vast number of subsequent pregnancies, it would seem unwarranted to assume that pregnancy alone is the factor which "lights up" the old latent infection. On the other hand, it is warranted to believe that a semiquiescent or slightly active lesion in one of lowered

resistance, in whom the warfare between the body forces and the infection is being carried on in the balance, might be definitely and unfavorably influenced by the increased demands made upon the body by pregnancy and labor. It is to be remembered that our wards contain the poor and hard working of the community, and for this reason the percentage may be higher than among the better class, as among poorer patients illness oftentimes does not make them consult a physician until there are marked symptoms or physical disability, and when these occur in pulmonary tuberculosis the disease is, as a rule, advanced. The date of onset as given by the patient is usually that of active tuberculous manifestations beyond the early stage.

The frequency of deaths from tuberculosis among women of the child-bearing age is very high. Bacon estimated, for example, that in the United States, yearly, 44,000 to 48,000 women of the child-bearing age die of tuberculosis, and that gravidæ and puerperæ furnish one-fourth of all the deaths from tuberculosis in women of this period of life.

The *problem* which I want to discuss with you today is our attitude toward the pregnancy. It is not how to treat the tuberculosis, because that is carried out along established lines, in or out of a sanatorium, as the conditions warrant. There is a difference of opinion with regard to our attitude toward the pregnancy, and our action will depend upon our conception of (1) the influence of pregnancy on tuberculosis and (2) the influence of tuberculosis on pregnancy. Let us consider briefly these factors.

#### INFLUENCE OF PREGNANCY ON TUBERCULOSIS

As previously stated, 30 per cent. of our married women patients dated the onset of their symptoms to a recent pregnancy or parturition. Various other observers have noted the large proportion of cases dating lung trouble to a pregnancy. Thus Jacob and Pannwitz found 25 per cent.; Fishberg, 37.4 per cent.; Maragliano, 59 per cent., and Trembley, 63 per cent. of the cases under their observation. A distinct aggravation of the disease was noted during pregnancy in 64 per cent. of cases studied by Diebel; 66 per cent., by Kaminer; 50 per cent., by Merletti, and

70 per cent., by Von Rosthorn. The course of the disease is usually progressive following parturition, and Bacon states that "one-third of the pregnant tuberculous women die within a year of their labor." There are a few observers who have had a different experience: thus Rabnow and Reicher report that 7 of 10 working women under treatment for active tuberculosis passed through pregnancy without signs of aggravation of the disease. Cohn reports 53 of 58 cases apparently uninfluenced by gestation; and Kohne thought the lung condition improved in 16 of 22 cases. These observers, however, are in the minority, and the majority of authorities are of the opinion that although a few cases of pregnancy and labor may seem to be without influence on the disease, nevertheless in the vast majority of instances the effect is distinctly unfavorable. A few isolated cases may seem to improve or be uninfluenced by pregnancy, only to rapidly decline during the periods of gravest danger; namely, labor and the lying-in period. It is important, therefore, in considering the influence of pregnancy to include not only the period of gestation but also the inevitable termination, parturition, puerperium, and lactation. The physical exertion, loss of blood, exhaustion of labor, and the occurrence of lactation, not to mention the demands made in abnormal cases, call upon the reserve of the patient, who in the presence of an active tuberculous infection is little able to respond without adversely affecting the pathologic process. Another danger to be reckoned with is that of auto-infection by aspiration of infected material from an old focus in the lung into healthy portions as a result of the violent respiratory movements during labor. Hanau pointed out this fact in 1887 in autopsies on tuberculous women who died soon after labor. He demonstrated, in addition to the old pulmonary lesions, new depositions of tubercles, which apparently could be explained only on this basis. The effects of parturition, therefore, are to be reckoned with in many instances more seriously than the effects of pregnancy. The latter assumes greatest importance when associated with nausea and vomiting, toxemia, or any abnormality. The view of the profession in the past, as expressed by Dr. Edward Warren in his prize essay of

1857, is no longer tenable. Dr. Warren wrote in part that "pregnancy, coition, etc., are particularly desired by women affected with phthisis, which constitutes a pointing of nature toward a remedy for the evils by which the system has been invaded."

#### INFLUENCE OF TUBERCULOSIS ON PREGNANCY

A history of sterility is uncommon in patients with pulmonary tuberculosis; indeed, it is a well-known fact that conception occurs frequently in patients with advanced disease. Marked anemia and marked general debility of an advancing lesion militate against conception, but in early cases its occurrence is rarely restricted. Among 100 of our married tuberculous women, 10 had never been pregnant. Of this number, one patient had been married only four months, and the effort was made to prevent conception, and one patient was operated upon four years prior to marriage for bilateral tubo-ovarian abscess. The husbands of the remaining 8 are said to be in good health, and, unfortunately, we have been unable to examine them with regard to their lung condition or their part in the childless marriages. The average duration of married life in this group of non-pregnant females was 10.6 years. It is interesting to note that in one of them, aged thirty-nine, and married at twenty-eight, the onset of the disease is dated from an operation performed under ether anesthesia one year ago for the correction of the existing sterility. Among the remaining 90 cases the average number of pregnancies was 4.7 per patient. Miscarriages occurred in 17 patients (18.8 per cent.) from one to four times, or computed upon the total number of conceptions, in 7.4 per cent. of the pregnancies. In 3 of the 17 patients the onset of the tuberculous manifestations was directly referred to a spontaneous abortion (ten weeks, one and a half years, and six years ago). In one instance of pregnancy terminated prematurely three times in succession, tuberculous manifestations occurring for the first time during first pregnancy, and becoming aggravated during and following each gestation. Among the remaining 13 the miscarriages antedate the onset of tuberculosis. May some of these miscarriages,

otherwise unaccountable, result from the toxemia of an early tuberculous lesion? Excessive coughing, progressing anemia, and high fever may be potent factors in causing abortion in some cases.

#### CONCLUSIONS AS TO THE ADVICE TO BE GIVEN A TUBERCULOUS WOMAN

It is our belief that the tuberculous woman should be advised against marriage and conception unless the lesion has, to all intent and purposes, been cured; that is, if the patient has been free from local and constitutional symptoms for a period of two or three years under ordinary conditions of life. If conception occurs our attitude with regard to it depends (1) upon the activity of the tuberculous lesion and (2) the stage of the pregnancy.

If the tuberculous lesion is early and active the pregnancy should be terminated if it has not gone beyond four and a half months. In such a case one should obtain the combined views of a chest specialist as to what constitutes an active or menacing lesion, and of the obstetrician as to the possible influence on the general condition of the patient by the termination of pregnancy. Unless the emptying of the uterus is deemed immediately imperative the safe precautionary method is to place the patient in bed, study the temperature and pulse, and observe closely the symptoms of pulmonary activity. If it is found that the lesion is active or threatens to become active the uterus should be emptied at once by a skilled obstetrician with as little trauma and shock as possible. The question of the anesthetic is to be decided by the operator, but it is the opinion of the writer that ether should be prohibited.

If the tuberculous lesion is early and inactive, for example, in a patient who gives a previous history of having undergone sanatorium care, but in whom the signs and symptoms are those of an arrested or cured case, the pregnancy should be left alone, particularly if, in spite of previous advice as to preventing conception, it had been disregarded. The patient ought to be placed under the best hygienic care, and a schedule of rest, ex-

ercise, and diet, with an abundance of fresh air, prescribed. If in doubt as to the inactivity of the lesion, the patient should be placed under close observation of the temperature, etc., as described in the previous paragraph. If under the best conditions the tuberculous lesion threatens to become active, the pregnancy should be terminated. An early inactive case, however, should be left entirely alone.

If the tuberculous lesion is early and active, and the pregnancy beyond the midperiod, we agree with Norris and Landis that little is to be gained by emptying the uterus, and it is best to allow these patients to go to, or nearly to, term, at which time labor should be made as easy as possible and special care directed to the patient during the puerperium, which is perhaps the most menacing time. Because the lesion is early it is often possible to gain considerable ground by placing the patient under immediate hygienic dietetic care. If not, the lesion may be kept fairly well under control till pregnancy is terminated prematurely. If the tuberculosis is so acute that the termination of pregnancy would seem immediately necessary, the chances are that such a termination will not help an already overwhelming lesion. In most instances such a patient will come to an untimely end, irrespective of operation.

If the tuberculous lesion is far advanced and the pregnancy likewise, a watchful waiting is all that is indicated.

#### THE CARE OF THE CHILD

If the pregnancy has been allowed to go to term, it will be found that unless the disease of the mother is far advanced children are frequently quite healthy at birth. Congenital tuberculosis is exceptional, and while various observers have reported instances of apparently transplacental infection, we must regard such infection as uncommon. The postnatal influence of tuberculosis in the mother is probably of greater moment to the child, because it intimately concerns the two essentials of its welfare: (1) proper nutrition and (2) wholesome environment. The first of these brings up the question of our attitude regarding nursing the child. Nursing in a tuberculous woman is

a menace both to the woman and to the child. Lactation is a demand upon the mother's nutritive reserve which is so necessary in the active combating of the disease. The abstraction of lime salts, so valuable in the pathologic process, occurs both during pregnancy and lactation (Lobstine). Nursing should be prohibited and, if possible, a healthy wet-nurse substituted or artificial feeding instituted. With regard to the child, it may seem undesirable to pursue this course, in view of the fact that the milk of tuberculous women rarely contains tubercle bacilli. However, there is a distinct danger of infection from other sources in the mother, a danger which is greater than one would suppose upon first thought. The soiling of the mother's hands with infective material from the mouth, the soiling of the breast in handling, etc., open many avenues for infection of the child; in fact, the way to reduce this danger to a minimum—the ideal way—would be to separate mother and child from ordinary contact. This may seem an exaggerated refinement, but it should be recalled that many have had the experience of Armand-Delille, who found in investigating the fate of the children of tuberculous families that among 787 children in 175 families, the 323 placed in the country by the Child Preservation Society are all well; of the 396 not helped by the society, 238 developed tuberculosis (over 60 per cent.).

Langstein states that children in the first and second years of life are from eight to nine times more liable to tuberculosis than adults. He quotes Pollack, who found among 285 children from families containing patients with open tuberculosis, 275 children with a positive tuberculous reaction. In this group of children it was found that clinical tuberculosis existed most frequently when exposure existed from birth; thus, among the 57 who were first brought into contact in the third year of life, only 7 showed clinical tuberculosis; among the 61 who were exposed in the second and third years, 45 showed clinical tuberculosis, with 7 deaths; and among the 207 who had been exposed to infection since birth, 200 showed clinical tuberculosis, with 21 deaths. The elimination of contact infection in infancy is one of the important problems of the antituberculosis crusade.



## CLINIC OF DR. MARTIN E. REHFUSS

JEFFERSON HOSPITAL

### MEDICAL TREATMENT OF BILIARY AFFECTIONS

WITHIN the last five years the medical treatment of biliary affections has undergone an important change. This change consists in a transition from systemic to local treatment; from empirical to specific treatment based on the newer anatomic and pathologic conceptions of biliary disease. The first concept which must enter our minds is the fact that the biliary tract,

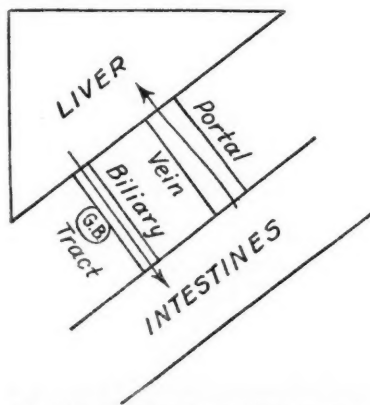


Fig. 145.—Intestinal hepatobiliary system.

the gall-bladder included, represents the connecting link between the liver and the intestines. In fact, in a broad way we have an afferent and an efferent system between these two units. The portal system represents the afferent system, transmitting the material from the intestinal laboratories to the complicated laboratory situated in the liver. The latter plant, apart from

its other functions, prepares the bile and sends it back to the intestine by means of the efferent tract. This conception emphasizes the importance of both hepatic and intestinal action in the production of biliary pathology. It becomes immediately evident that infection of the biliary passages, apart from the hematogenous infections, must take place either (1) by hema-

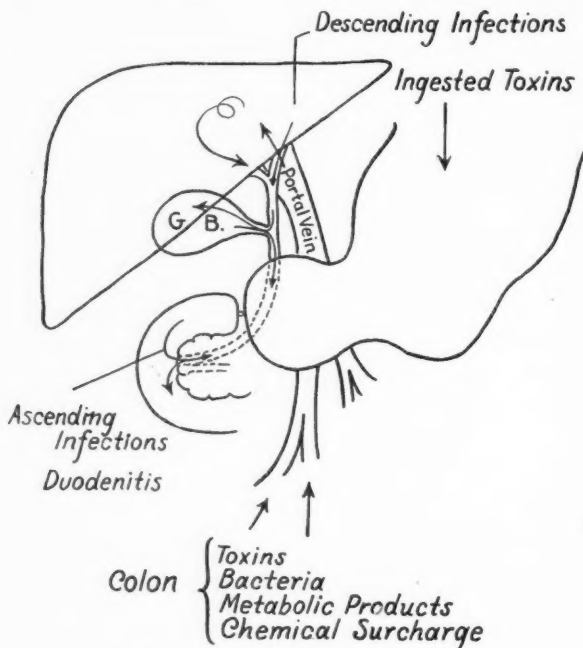


Fig. 146.—Methods of biliary infection.

togenous infection from foci elsewhere, (2) from ascending infection (from the intestines), (3) descending infection from the liver. The possibilities of alteration in the chemical composition of the bile as well as its bacteriologic contents are only becoming manifest as this subject is being more thoroughly investigated. Two points stand out clearly: first, that there are distinct alterations in the chemical composition and viscosity of

the bile, such as the increase in the cholesterol content favoring cholelithiasis, and alterations in the pigments, salts, the urobilin, and urobilinogen, as well as the calcium salts, the exact significance of which are not as yet fully known. On the other hand, the presence of pathogenic organisms and particularly the elimination of organisms of the typhoid colon group is assuming increased importance as newer studies indicate its frequency. *We are gradually working around to the idea that there is not merely an elective localization of pathogenic organisms, but, as Richet pointed out, there is also an elective elimination of these same elements.* If organisms of the dysentery group are eliminated by the large bowel after injection into the blood-stream, it is equally true that organisms of the group mentioned above are most frequently eliminated by the hepatobiliary tract.

On the other hand, the association of duodenitis, concerning which we are obtaining more and more information, and more often gastro-duodeno-enteritis, makes it probable that ascending infection is not rare despite the fact that there is at times a positive pressure in the gall-ducts. These conceptions introduce as factors in biliary disease nearly the whole gamut of hepatic and duodenal conditions. This is consistent with the fact that many advanced hepatic disturbances are unaccompanied by evident biliary change, the inference being that the bacterial or chemical alteration is not sufficient to induce demonstrable change.

The mechanism of infection demands some mention in these conditons. Certainly the most common infecting organisms are the colon bacillus, streptococcus and staphylococcus, and the pneumococcus. It is probable (1) that the three last mentioned organisms are usually hematogenous, and the first represents the type most frequently in association with obvious intestinal disturbances; (2) many diseases are essentially polyvalent in their bacteriology, as, for instance, the association of the influenza bacillus, streptococcus, and pneumococcus in the recent epidemic of grip, and the association of the streptococcus in many cases of pneumonia. These polyvalent infections are frequently accompanied by the implantation of one or more

organisms showing an elective affinity for this region; (3) the alteration in virulence of chronically infecting organisms producing the association of appendicitis and cholecystitis along the lines laid down by Rosenow.

I would mention a new factor which to my knowledge has not been emphasized, but which from my observations occurs with great frequency. I refer to the association of intestinal stasis and a definite deviation from normal in these cases. The action of intestinal stasis is far from completely understood. It is not simple stasis, with the absorption of toxic products, but far more important are the changes in intestinal rhythm which follow this advent. Aërophagia, heart-burn, globus, coated tongue are not merely symptoms attending intestinal stasis, but symptoms produced by a mechanism which Alvarez has referred to as "mid reversed peristalsis." *There is no principle recently enunciated in gastro-intestinal pathology which is more important than the various modifications of reversed peristalsis.* Our studies in the Jefferson College on the mechanism of duodenal regurgitation were but a small amount of the evidence accumulating in our sight to suggest the ready manner in which normal continuity and rhythm can be markedly changed. It is probable in every case of high intestinal stasis that a definite degree, more or less marked, of reverse peristalsis occurs, resulting in the regurgitation of jejunal and ileal contents back toward the duodenum. In fact, in almost 80 per cent. of all biliary cultures taken by us according to the method mentioned, the colon bacillus was found (a possible explanation to me being mild reverse peristalsis with implantation of the colon bacillus high in the small bowel). This is not normal, inasmuch as we encountered negative cultures with sufficient frequency, but the above figures were obtained in diseased conditions. One explanation is the elimination of the colon bacilli, the other is the mechanism mentioned above. I have repeatedly demonstrated that relief of high intestinal stasis through high intestinal irrigation results in a disappearance of the symptoms.

Recent anatomic and physiologic studies support the contention of Meltzer that there is a mechanism of contrary innerva-

tion similar to the urinary bladder in which the smooth muscle of the gall-bladder and the Oddi muscle at the termination of the common bile-duct play the reciprocal parts, contraction of one resulting in dilatation of the other, so that when the Oddi sphincter is contracted and the gall-bladder relaxed the organ fills up, and vice versa. The efficiency and rhythmicity of this function depends on its efferent stimuli, balanced dietaries and regular eating producing regular and complete evacuation, irregularities in eating resulting in undue stasis of bile.

It therefore follows that, apart from neoplasm and contiguous diseases, disease of the biliary passages arise from three sources: the blood-stream, the lymphatic stream, and the bile, and the bile undergoes three separate forms of alteration: infection, stasis, and alteration in its chemical constituents. Infection takes place through the blood-stream, the lymphatic channels, ascending from the bowel and descending from the liver.

#### DIAGNOSIS OF BILIARY DISEASE

In the diagnosis of biliary disease the internist is armed with a new and valuable procedure in the shape of duodenal intubation. In this way the bile can be studied directly as is the gastric secretion, and the various forms of chemical analysis can be applied to the material removed. In other words, we can make an examination of the bile from the standpoint of its chemical constituents, namely, cholesterol, bile-pigments, and salts, urobilin and urobilinogen, calcium content, as well as the determination of viscosity; from the microscopic examination the presence of mucus, pus, blood, and laminated cholesterol plates; and from the bacteriologic examination the determination of the presence of pathogenic bacteria. Inasmuch as all these procedures depend for their determination on an accurate method for obtaining bile, the following details are given: the patient is examined the first thing in the morning upon an empty stomach. After preliminary rinsing of the mouth with some antiseptic solution, the long, fractional gastroduodenal tube is passed, according to the ordinary technic, for 70 cm. or more. Ordinarily I pass a length of tubing sufficient to go from the

mouth, externally, to the anterior superior spine of the ileum. The tube is usually passed with the aid of beef-broth or bouillon which has been sterilized by boiling. If the broth is given at the same time that the tube is swallowed the passage of the instrument is materially facilitated. Occasionally the tube will be found to enter the stomach more readily after milk is ingested, the digestion of the curds apparently favoring the passage of the tip. As soon as the tube is introduced the patient is turned on his right side with the left leg drawn up and thrown over the right in order that the pelvis be definitely rotated in that direction. The lapse of time before the tip enters the duodenum varies with every individual; in fact, there is no method by which we can predict the duration of time necessary to intervene before bile is obtained. The bile is readily recognized by its appearance, and it will be noticed that the flow is intermittent. In obtaining bile cultures the following procedure is the one which has recommended itself to us: after the bile is obtained the patient is put in the fully recumbent position with the head directed toward the edge of the bed, the remaining 2 feet or more of tubing is allowed to remain suspended over the side of the bed in such a way that by siphon action there will be a tendency toward a steady flow of bile. After the flow has commenced and the first part is thrown away, three culture-tubes are used in the following way; the first tube is placed under the drip without allowing the tube to come in contact with the fractional tube, and after the first culture of bile is obtained the second culture-tube is used in the same way, followed, in turn, by the third. This method has its advantages and its disadvantages, which are readily apparent even upon superficial observation. The disadvantages are the following: it is difficult to maintain the sterility of the tube owing to the fact that its transit into the duodenum must necessarily lead through organs which can be only incompletely sterilized; second, the bile is not usually obtained in a pure state, being associated with the pancreatic secretion, the succus entericus, and not infrequently some of the gastric contents. This means that infection in this region can be also ascribed to a duodenitis.

On the other hand, the following points can be claimed for the method: after the initial flow of bile the material obtained is remarkably uniform, as are the resulting cultures. It therefore follows that external contamination will not give a uniform appearance in the various culture-tubes. Accidental contamination is recognized by the presence of isolated colonies and the lack of uniformity in the appearance of the three culture-tubes. A true bile infection is recognized by the fact that all three tubes have practically the same aspect and contain the same bacteria. We know of no way by which organisms coming from pancreatic secretion can be dissociated from those seen in the bile, and for diagnostic and therapeutic purposes there is no pressing need for such a differentiation. We have encountered the streptococcus and pneumococcus only infrequently; the colon bacillus, however, was far more apparent. Any form of media can be used. As routine procedures we employ blood-serum, agar, and bouillon. The vaccines are made in the ordinary way. The frequency of the colon bacillus has not yet been satisfactorily explained, whether it represents a form of elimination on the part of the liver into the bile; whether its presence is due to reverse peristalsis from the small bowel, as seen in the ascending or cecal type of constipation; or whether it represents a true infection, we are not prepared to state. Certainly the presence of abundant colon colonies is not a phenomenon of normal bile, nor is their presence in the upper duodenum to be associated with perfect health.

#### THERAPY OF BILIARY DISEASE

In a study of the therapy of biliary disease the following means are at our disposal:

**Specific treatment** can only be used in the presence of specific etiology. Vaccines constitute a specific form of therapy if we have isolated the causative organism or organisms. The above method offers us this possibility, and in every case culture of the bile should be carried out with the idea of preparing vaccines. Of great interest is the communication of Bertholet on the

method of isolation of intestinal organisms by means of amino-acid media. This whole subject, however, is too recent to mention in gall-bladder work. Kelly (Stitt), in a bacteriologic study of 413 operations on the biliary tract by Deaver, found the bile sterile in about one-half the cases, but in acute disease pathogenic organisms were almost constantly present. *Bacillus coli* was found in 30 per cent., *Bacillus typhosus* in 7 per cent., *Staphylococcus pyogenes* in 2.9 per cent., streptococcus in 0.2 per cent.; 55 per cent. were sterile. By the method which we use the percentage of positive cultures is higher, but the relative incidence of other organisms about the same. Stitt mentions that in one case of cholecystitis the colon bacillus found was markedly hemolytic on blood-agar plates. There is no question but what there is a marked difference in the virulence and pathogenic properties of various colon organisms, and also a fact which I have noted, namely, that an autogenous vaccine from the colon bacillus isolated from the bile gave in one case of mine much more pronounced reactions than the same organism isolated from the feces (*arthritis deformans*). These differences make us hesitate to say whether a colon growth is due to colon organisms normally eliminated through the liver, or whether it is due to infection of this region. As I have already pointed out, the cultures are taken by the drip or siphon method; in order that an organism be used, it must either occur in pure culture or in sufficient frequency on all of the three tubes to be used as a vaccine.

We begin with usually 25,000,000 of the *Bacillus coli*, 100,000,000 of the ordinary pus organisms. Occasionally unusual organisms are encountered. For instance, on three occasions (three separate examinations) we encountered in pure culture in each instance in an old case of angiocholitis, repeatedly operated on and complicated by many adhesions, the *Bacillus pyocyaneus*. In several instances we got a pure growth of the pneumococcus. The streptococcus, according to my studies, is very rare, although I have frequently used blood-serum. Sensitized vaccines are undoubtedly of value, but I have not used them in this work.

The following points are of value in this work:

(1) Nearly always the patient with gall-bladder or duct disease has foci of infection elsewhere. We therefore culture the nose, throat, teeth, frequently the urine, and when a definite strain is located in the gall-bladder, we make a polyvalent vaccine consisting of not merely the organism isolated from the bile, but the same organism found elsewhere. A difference in virulence undoubtedly occurs, as mentioned above, and this method contributes to the likelihood of an effective antigen.

(2) Autogenous vaccines are to be preferred to stock vaccines, but the presence of organisms elsewhere which are persistent and suspicious in the mind of the clinician ought to be included. I believe that the good results I have obtained have almost always been due to this precaution. Only rarely do we give the single organism.

(3) Small, frequently repeated, and increasing doses are given until a definite reaction is obtained. Strong reactions are to be avoided, although some of the best results that I have obtained have been following a strong reaction with active temperature rise, and severe general reaction. In general, however, only those vaccines do good which give a reaction, but a dose should be used which provokes only a slight reaction. A strong reaction means reduce the next dose. In fact, there is no rule as to the amount of vaccine. If about four steadily increasing injections are given without reaction, the vaccine treatment is discarded. In some cases there is a distinct reduction in the virulence or growth ability of the organism without their disappearance, a point which I have noted on several occasions.

(4) Occasionally definite organisms are found, vaccine prepared and given, but, in spite of reactions, no improvement occurs. The following is such a case:

Mrs. H. Subicterus, bile in urine, tender gall-bladder; repeated attacks pronounced intestinal stasis and general ptosis. Cultures gave pronounced growth of colon and *Staphylococcus pyogenes* on three successive occasions. This patient received cautiously vaccines (autogenous), with no improvement. Operation disclosed stone in common duct and in gall-bladder and

chronic cholecystitis and angiocholitis. The gall-bladder when opened gave the characteristic indol odor of the colon organism. Here vaccine therapy naturally failed to induce a change, owing to the fact that it did not reach the active foci contained in the stones. It is questionable likewise whether vaccine therapy alters the ability of bile to maintain living colon organisms.

A second case represents a different phase of this problem. A. I., a young girl, had six operations on the gall-ducts by various surgeons in an attempt to relieve the pain and infection. Each operation made the duct more difficult to find, and on each occasion many adhesions were demonstrated. This is the case with the pure culture of the *Bacillus pyocyaneus*, and vaccines failed to relieve her in the slightest. It is probable that avascular tissues like scar tissue may harbor infection and prevent the full influence of immune bodies circulating in the area in response to vaccine injection.

The first of these two cases indicates the difficulty of dealing with a cholelithiasis and cholecystitis case. Almost always these cases are surgical, but the problem which the surgeon has failed to answer for us is the following: *Does drainage with or without cholecystectomy result in cure of the infection?* While it is true that numberless cases attest to the value of the operative procedure, it is equally true that in many cases a persistent angiocholitis follows. The following are cases of this description cured by vaccine therapy:

Mrs. C., mother of two children. Operated on for cholelithiasis and cholecystitis; the gall-bladder was removed. A year later there were still symptoms, and culture revealed both colon and staphylococcus. A mixed vaccine, with organisms from the throat containing staphylococcus, streptococcus, and colon, was given. At the time this treatment was commenced the patient was jaundiced. She received both local treatment, by means of the Murphy drip, and duodenal tube and vaccine and dietetic treatment, with a total disappearance of all symptoms.

Mrs. L. Operated on for gall-stones, had persistent tenderness, and symptoms referable to the gall-bladder region. A pure colon culture was obtained. This, together with organisms

from the mouth and throat (staphylococcus and streptococcus), was used. After eight injections, diet, and treatment of the bowel all symptoms disappeared.

These cases are illustrative of a series which on some future occasion I hope to assemble, but on their findings I base my belief that in the absence of many adhesions or stone vaccine therapy is of value in demonstrated infections of this tract.

**Dietetic Treatment.**—Best known among the forms of dietetic treatment for diseases of the biliary tract is the so-called low cholesterol diet in cholelithiasis. This is based on the definite conception that an increase of circulating cholesterol plays a part in gall-stone formation, and also on the fact that an increase in circulating cholesterol may be exogenously due to a diet rich in these substances. It therefore follows that cholesterol-rich substances are eliminated from the diet. Chauffard gives a list of cholesterol-low foods to be used in this condition. They exclude yolk of eggs, peas (phytosterol), fatty meats, and all fried foods.

I have used such a method, and while my surgical brethren do not agree with me, I can point to a number of individuals who undoubtedly harbor gall-stones (some demonstrated by the x-ray) who are free from symptoms. I am fully aware that when a stone migrates, no cholesterol-low diet in the world will relieve them. *Any method, however, which probably reduces further formation of stone is indicated in cholelithiasis.* I am also fully aware that by no means all stones are cholesterol stones. A fact which Rosenbloom recently pointed out is of value, namely, *that probably most cholesterol stones are non-infected, and that in the absence of demonstrable infection, as demonstrated by culture, this method of treatment is justifiable.*

Apart from the cholesterol content, the diet in these conditions should be low in protein and low in fats. *Low in protein because, as Meltzer pointed out, peptone is probably the best choleragogue, and low in fats because a high fat diet favors migration of stone extension or activity, and activity rather than rest to the diseased organs.* The formula, then, is high carbohydrate, low fat,

and low protein; such a diet is the classic diet of the French for disease in this region.

**Local Treatment.**—Direct treatment to the gall-bladder and bile-passages is impossible. Two means of treatment are possible: (1) To the ampule of Vater through the duodenum; (2) alteration of bile by altering the portal blood through the intestinal tract. I have used the *duodenal tube connected with*

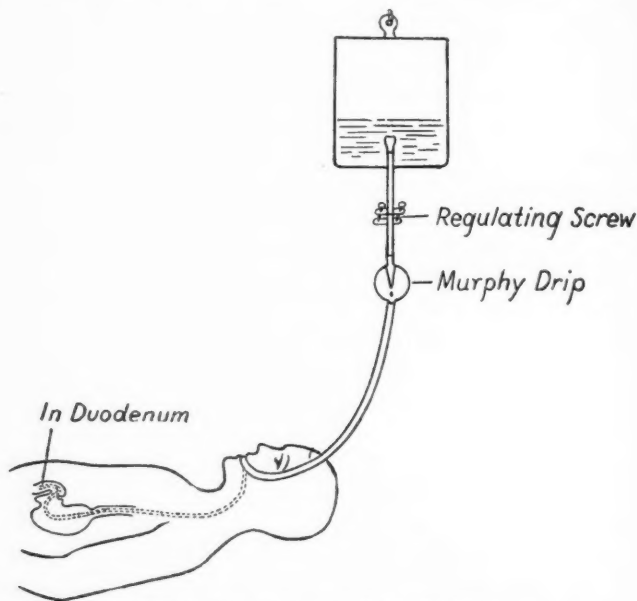


Fig. 147.—Drip system applied to duodenum.

*the Murphy drip.* This method of treatment consists in the passage of the duodenal tube or the gastroduodenal tube in the customary way. After bile is obtained, the end of the tube is linked up with the Murphy drip apparatus, and the disinfecting or cleansing solution run through at a rate of from 40 to 60 drops a minute. Solutions are used according to their purposes. Disinfecting solutions consist of the use of dilute solutions of the various silver solutions. I have used argyrol, protargol, and

even nitrate of silver, although the effect of the latter cannot be properly controlled. Methylene-blue has also been used in this way, as well as salicylic acid. As a rule, disinfecting solutions should be used in rather concentrated form, and usually I use only from 4 to 6 ounces of fluid in this way. Urotropin can likewise be used in the same way. I have used as much as 4

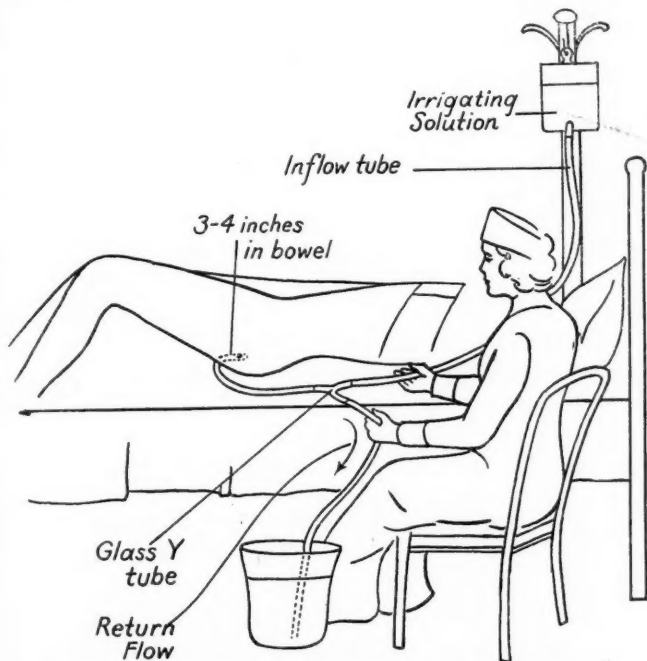


Fig. 148.—Nurse conducting double irrigation of the bowel.

grams at a single treatment. The second group of solutions are, in the main, cleansing and mucus-dissolving in their action. This includes concentrated solutions of sodium bicarbonate, sodium phosphate, sodium sulphate, or fairly strong dilutions of liquor antisepticus alkalinus. The laxative or cathartic effect can be obtained by adding these substances. I have treated many cases of infected jaundice and duodenitis in this way.

Some of them have been associated with cholecystitis and cholelithiasis. In the hands of an experienced nurse this treatment is often very successful.

The second point which impresses me as of great importance in these cases is the necessity of caring for the colon. Practically all of these cases have disturbances in bowel functions; in fact, the more I see of gall-bladder disturbances and also troubles with the gall-ducts, the more I am impressed with the primary importance of the bowel. The clinical symptoms apart from those which are purely localized are those associated with the "syndrome of mild reversed peristalsis." Acute disturbances are almost always associated with an increase in the irritability of the abdominal sympathetic plexuses and spastic tendencies in the bowel wall. Many are associated with evidences of colitis and enterocolitis; some infected others in which no actual influence of infection can be elicited. It must be evident that colon stasis and infection is followed by the transmission of these products by way of the portal system to the liver. The liver then must suffer, and its function be altered or perverted. Therefore it is logical to assume that modification of bile and of liver function should occur. *It follows that the logical treatment of hepatic and biliary disturbances should be by way of the bowel, which will directly or indirectly influence these organs by way of the portal system.* The influence of high irrigation of the colon and the marked improvement which occurs in certain cases must undoubtedly occur in this way. In almost every case it has been my practice to determine the condition of the colon, and to attempt to influence hepatic and biliary function in this way.

## CLINIC OF DR. J. P. CROZER GRIFFITH

UNIVERSITY HOSPITAL

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### DILATATION OF THE COLON IN CHILDREN, WITH ESPECIAL REFERENCE TO THE IDIOPATHIC FORM

DILATATION of the intestine, and especially of the colon, is frequently observed in early life. It may be divided into (1) the secondary and (2) the primary or idiopathic form; the latter being of unusual occurrence, but of great interest on account of the necessity of distinguishing clearly the common secondary form from it. The causes of secondary dilatation are various. In rare instances an organic obstruction exists, such as a congenital stenosis in the course of the lower colon or the rectum, or the pressure of a tumor from without upon the intestinal wall. Chronic constipation may eventually produce great dilatation of the colon, although this is a result oftener seen in adult life than before this period. Any long-continued debilitating disease may give rise to dilatation. Most marked in this class of causes is rachitis, and the abdominal distention produced by this is of every-day observation. The dilatation may occur in any part of the gastro-intestinal canal, but appears to be oftenest in the colon. The very great degree of atony of the muscles of the abdominal walls, as well as of those of the intestine itself, readily permits of dilatation developing, and this is aided, too, by the intestinal indigestion which is often present. Sometimes the food employed has been of a nature which easily favors fermentation in the intestinal canal, and consequent increase of its lumen. Whatever the cause, it is safe to say that more or less dilatation is found in every case of rickets, and that in the severer ones it is one of the most prominent features (Fig. 149). The

abdominal distention in many of these cases is rendered still more striking by contrast with the narrow, contracted chest which such subjects exhibit, the deep Harrison's groove, and the flaring costal border.

Another cause of persistent and often very considerable dilatation of the colon is chronic intestinal indigestion, as it occurs



Fig. 149.—Severe case of rickets with much distended abdomen. From the Children's Hospital of Philadelphia.

especially in the second year of infancy and during early childhood, *i. e.*, from the second to the sixth year. The small intestine is dilated as well, but the chief distention occurs in the colon. The general symptoms of this disease are more or less characteristic. There is a loss or perversion of appetite, anemia, irrita-

bility or other signs of nervousness, loss of strength, emaciation, disturbed sleep, and, it may be, retardation of growth. The bowel movements are in some cases constipated; in others loose, or these conditions may alternate. They may contain large amounts of mucus, or may be much too light in color, owing to the failure of the proper digestion of the fat of the food. Even when not of this color, microchemical examination may show an excess of this element. Undue fermentation of the carbohydrate portion of the diet takes place and results in the accumulation of a large amount of gas, and often in the production of irritating diarrheal evacuations. Various other manifestations, especially those of a nervous nature, are observed; but the most important symptom from the present view-point is the distention of the abdomen, which is nearly always present in cases past the period of infancy. The same causes are operative here as in rachitis; namely, the atony of the abdominal and intestinal musculature, and the abnormal intestinal fermentation. In many instances the abdominal distention is by far the most striking feature.

Another cause of abdominal distention in early life is pneumonia. Here the condition is of an acute nature, and probably the whole gastro-intestinal canal shares in the distention. Little further reference need be made to it here, except to point out its bad prognostic indication. It probably results from the paralyzing action of the toxemia of the disease, and not only indicates the severity of the attack, but itself adds to the distress and danger by the upward pressure of the gas against the diaphragm, and the consequent interference with the action of the heart and lungs.

Acute peritonitis may produce a temporary dilatation of the small intestine and colon through the paralysis of peristalsis; and tuberculous peritonitis may give rise to a gaseous distention of a much more chronic nature.

The form of dilatation of the colon, however, to which especial reference is made in this connection is often denominated congenital idiopathic, and is sometimes called Hirschsprung's disease. The following is the history of an instance of this interesting disorder:

William W., aged three years and four months. The child was brought to the Children's Ward of the Hospital of the University of Pennsylvania September 3, 1918, on account of vomiting and chronic constipation. The mother said he had been subject to attacks of vomiting since birth, especially at times when purgatives, such as castor oil and Epsom salts, had



Fig. 150.—Idiopathic dilatation of the colon in a child of three years and four months in the Children's Medical Ward of the Hospital of the University of Pennsylvania. The peristaltic movements can be seen below the left costal border.

been administered to relieve the constipation. At such times he lost his appetite, but, as a rule, this was good. The attacks of vomiting were said to have lasted a few days at a time or even a week. The vomiting caused but little discomfort. There had always been constipation, and a week passed at times without an evacuation. The feces were said to be dark-colored and lumpy, and on occasions to have contained large curds. The time when

the distention of the abdomen was first noticed was not stated. The child had been in good general health, of fair weight, playing out-of-doors, and eating and digesting well, except during the attacks of vomiting.

*Examination on Entrance.*—The patient is rather apathetic. The general health seems good. The head is slightly rachitic in shape. The tonsils are enlarged and the mouth is held open a portion of the time. The chest is only fairly well developed, but is without rachitic rosary. The most marked feature is the very large abdomen, which is tympanitic on percussion, and exhibits, especially when manipulated, very active peristaltic movements of large excursion, the outline of the much distended colon showing distinctly through the abdominal wall (Fig. 150). The peristaltic movements of the small intestine are also visible. The arms and legs are thin, but otherwise normal, except for a moderate degree of genu valgum.

*Later History While in the Hospital.*—The child grew very bright and playful, with abundant vitality, as he became acquainted with his surroundings. A test-meal given showed nothing abnormal in the digestive or propelling power of the stomach. Constipation remained a marked feature unless relieved artificially. The muscular wall of the abdomen was decidedly thin and weak, tense, and with a slight umbilical hernia. The forcible waves of peristalsis persisted, and were attended by a very audible gurgling sound. A fluoroscopic examination was made and x-ray plates taken after 3 pints of a suspension of barium had been given by rectal injection. There was hesitation about administering a larger amount, as this quantity was as great as would have been used in the case of an adult. The enormously distended colon could be readily seen (Fig. 151), but the amount of fluid used did not extend as high as the transverse colon. The rectum was not distended. Measurement of the abdomen at the umbilicus gave a girth of 23 inches (57.5 cm.). Vomiting occurred for the first time on September 18th, fifteen days after admission, and then only once. Massage of the abdomen was given daily and the diet regulated to contain as little waste material as possible. Liquid petrolatum was

administered by the mouth and soap enemata employed. About September 30th the patient was attacked by the epidemic of



Fig. 151.—Radiograph of Fig. 150, viewed from behind. Showing the great distention of the ascending colon. Not sufficient fluid injected to pass into the other parts of the colon.

grip then prevailing in the hospital and elsewhere. During this there was a return of vomiting, and diarrhea developed, and the child became apathetic and quite debilitated. The diarrhea

persisted for some time, but without any special diminution in the girth of the abdomen. Later the severe symptoms disappeared, the patient reached his former bright and happy condition, and he was sent to the country to convalesce, where he is at this writing.

Idiopathic dilatation of the colon is an affection even yet not common, although more often recognized than was formerly the case. In an article published in 1889, which included the description of an instance of the disease, I could collect but 23 previously published reports which seemed without question to belong to this category. Since then the number of recorded cases has been increased greatly. Finney, in 1908, gave a list of 206 publications upon the condition; Patel, in 1910, collected 223 cases, 200 of which had been confirmed by operation or by post-mortem examinations; and Porter and Weeks, in 1915, added 100 more to the list. While it is true that Hirschsprung in 1888 made the first careful, analytic study of the disease; yet instances had repeatedly been reported earlier, as by Parry, Billard, Henoch, Peacock, and Hughes. There seems no good reason, therefore, why the disease should bear the name of Hirschsprung rather than of another.

The nature and cause of the malady are still far from being thoroughly understood. There probably exists a congenital tendency for the colon to dilate, although the actual dilatation is not always present at birth. On what the tendency depends and in what manner the dilatation is brought about are by no means clear. The disturbance has been supposed to be neuromuscular in nature, being a paralysis of some region of the colon with arrest of peristalsis; or perhaps a spasm producing functional obstruction; and still other views have been entertained. These theories have not been satisfactorily confirmed by post-mortem examinations. The dilatation is clearly not the result of a general muscular atony, for, although this is operative in that developing in severe rickets, the subjects of idiopathic dilatation are children in other respects entirely healthy. Much has been written of the influence of the remarkable length of the colon, and especially of the sigmoid flexure, characteristic of early

life, as compared with the rest of the intestine; but the very fact that this condition is so very common is immediate proof that it cannot be more than a predisposing cause of dilatation, as otherwise the disease would be of every-day occurrence. It has been maintained, too, notably by Treves, that the dilatation may depend upon some constriction in the lower part of the colon or in the rectum. Such cases exist, and I have seen one interesting example in which the diagnosis was made by operation, and recovery followed. But it is clear that any such cannot be called "idiopathic"; and autopsies have failed to reveal any condition of this kind in the majority of instances. It has further been a matter of dispute whether the hypertrophy of the intestinal wall, which is found as a nearly constant occurrence, is a cause or a result of the dilatation. Certainly in some fatal cases in early infancy both conditions have been present, and in these the hypertrophy could not have been a secondary process depending upon constipation. We are not justified, therefore, in assuming that the constipation is the necessary principal factor which is finally followed by dilatation of the lumen of the bowel and hypertrophy of the walls.

The lesions found at autopsy are characteristic. The colon may be involved throughout, or only the sigmoid flexure. In some instances the rectum and the lower part of the ileum are also affected. In an analysis of 169 cases published by Neugebauer the sigmoid alone was dilated in 74, and the entire colon in 32; in 26 the rectum also was involved. The distention is frequently so enormous that on opening the abdominal cavity little other than the dilated large intestine is visible. In addition to the dilatation there is nearly always found thickening of the intestinal wall. This produces a lack of elasticity in it and increases the trouble. In long-standing cases inflammatory changes and even ulceration develop.

The symptoms need be but briefly discussed, since they are so characteristic. They are principally two—constipation and great distention of the abdomen due to the dilatation of the colon. Usually constipation is the first of these to appear, followed more or less promptly by the other. The symptoms may be present

at birth, but in the large majority of cases (153 of 210 cases collected by Neugebauer) only the *tendency* exists at this time. The symptoms themselves generally appear in the first three months. Occasionally actual dilatation does not develop until the age of a year. The constipation is of a most obstinate nature, the child passing one or two weeks, it may be, without a stool, unless relieved by treatment. A stool without assistance is uncommon. After such an interval the evacuation is, of course, enormous. It is noteworthy that the fecal matter, although often dry and in large masses, is not, as a rule, scybalous, the constipation depending not upon any characteristic of this nature, but upon the lack of expulsive power of the intestine. In fact, in some instances, at least for a time, as in the case now reported, the stools are diarrheal in nature, and there may be for periods a liquid evacuation daily, yet without necessarily any great difference in the degree of abdominal distention. The amount of distention, in fact, varies with the case, and to some extent from time to time in the same patient. After a thorough evacuation by enemata there may be quite a considerable reduction in the girth; but this is not true in every instance. In the case now reported the circumference of the abdomen equalled 23 inches (57.5 cm.); and I have seen other instances in children younger than this patient with a greater girth. In cases not too far advanced very active peristaltic movements of the colon are visible. This was very markedly true of the instance here recorded. It is remarkable how comfortable and in what good general health the patient may be for a number of years. There is seldom much pain, and vomiting cannot be regarded as a characteristic symptom. Dyspnea may be produced by the pressure upward against the diaphragm, but, as a rule, not to any great degree.

The ultimate prognosis is not favorable. Of 24 cases of my own previous report, 18 were known to have died. In 59 cases treated medically, as recorded by Löwenstein, the mortality was 66 per cent. In 110 operated cases collected by Terry the mortality was 44 per cent. It is not usual for the subjects to reach adult life, and most of them die before the age of five years from

increasing debility or from some intercurrent disease. Treatment is, therefore, a matter to be considered with care. To operate would at first sight appear to be the safest course, doing by preference a resection of the dilated portion of the intestine; but in view of the high mortality which has attended the procedure it seems oftener wiser to temporize, if the general health is not being impaired. In 2 instances seen within the last two years I have advised delay. One was a vigorous colored child of four years, whose case has been reported elsewhere; the other the subject whose history we have been considering. In each instance the general health was good and the patient in no way suffering through the delay. In such instances relief may be obtained from time to time by colonic irrigation or by purgatives. These procedures should not be resorted to oftener than necessary, as there is danger of increasing the weakness of the expulsive power. Strychnin may be administered in the hope that it may increase the intestinal tone, and abdominal massage is certainly useful in aiding the expelling of the intestinal contents.

After all, however, the practical importance of idiopathic dilatation is inconsiderable by itself, inasmuch as the disease is so seldom encountered. What *is* of the utmost importance is the diagnosis between this and other forms of dilatation, since should this not be clearly established and should operation be advised in cases which are of a purely secondary and remediable nature, the patient is placed in entirely unwarranted danger of losing his life through the mistake of the physician. For this reason the discussion of diagnosis has been deferred until now. I recall with vividness an instance in which I was called in consultation to confirm the attending physician's opinion that the disease was idiopathic dilatation of the colon, and to sustain him in the advice he had given that the colon be resected. The patient in the condition in which I then found him might with good reason have been considered a case for operation so far as the symptoms present at this time were concerned; but it so happened that the little boy, then four years old, had been under my own care during the first two years of his life, and that I knew he had been the subject of severe chronic intestinal indigestion with abdominal

distention. This enabled me to make a diagnosis which might otherwise have been difficult. The little fellow was subjected to no operative interference and is now an entirely healthy child.

The diagnosis of the nature and cause of dilatation of the colon is, as intimated, by no means always easy. The acute abdominal distention attending pneumonia is readily recognized, and should be so promptly, on account of its bad prognostic import. It calls for all possible measures to sustain and increase the vital forces. In occasional instances where other means for obtaining an expulsion of the gas have failed, I have seen the hypodermic injection of eserine act most satisfactorily, giving  $\frac{1}{500}$  to  $\frac{1}{300}$  grain at the age of two years, and repeating this if necessary. The drug is, however, a possible depressant and may do damage, although this may be counteracted to a certain extent by combining it with strychnine. The gaseous abdominal distention seen in acute peritonitis is, as a rule, readily recognized by the accompanying symptoms; that attending some forms of tuberculous peritonitis is not so easily distinguished. Time, however, will solve the matter. The onset in tuberculous peritonitis is not so slow, and the condition occurs later in life. There is abdominal tenderness, and fluid or tumor masses may be discovered, the latter being distinguished from fecal accumulations by their permanent and fixed position, not affected by colonic irrigation. Fever is present, and there are generally other evidences of tuberculosis. The general health is usually early affected. It should be stated, however, that in some cases of idiopathic dilatation fecal masses felt through the abdominal wall may not be removed by enemata.

Rachitic dilatation may be very decided and very prolonged, yet can rarely cause any difficulty in diagnosis. It develops in infancy, it is true, but only after earlier symptoms of rachitis have shown themselves; and it is not as great as in idiopathic dilatation. It is attended by contracted thorax and general poor health without obstinate constipation. The prognosis is, of course, good, so far as the distention is concerned, the general rachitic condition being that which needs immediate and prolonged treatment.

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The numerous cases of chronic intestinal indigestion accompanied by distention of the abdomen are those which are most likely to cause errors in diagnosis. This disease begins usually at a later period than idiopathic dilatation of the colon; bad health precedes the dilatation, there having been long-continued intestinal symptoms, especially diarrhea; constipation is never as pronounced; the distention is usually not so great. It is, in fact, difficult to confound the two if a satisfactory previous history of the patient can be obtained. Operation is, of course, never indicated in this disease. Recovery, though slow, is to be hoped for in most instances through care for the regulation of the hygiene and diet. The nature of this is a matter too extended to be discussed further in this connection.

## CLINIC OF DR. MAURICE OSTHEIMER

### UNIVERSITY HOSPITAL

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#### FEEDING BABIES DURING THEIR SECOND YEAR

THIS baby is shown to you simply as a text. You will all note that she is not sick—on the contrary, she shows all the marks of an exceptionally well child. You can definitely place her among the “overfed” at once, from her looks alone. Let me give you the few important facts in her history. She was breast fed for only three months; then her mother weaned her for no real reason whatever. She was given condensed milk, then malted milk, then the various other proprietary foods. We first saw her when she was six months of age, a pale, flabby, undernourished infant, weighing  $10\frac{1}{2}$  pounds. It took little time and very little trouble to start her on a cows' milk mixture, 6 ounces every three hours, slowly. She improved at once, and her mixture was gradually increased until she finally reached whole milk at eleven months. At one year she had five teeth, could chew zwieback well, took oatmeal, orange-juice, and prune-juice once a day besides her milk. She weighed 20 pounds at twelve months of age.

She is now twenty-five months old and weighs 33 pounds. Her appetite is excellent, but she gets only three meals a day, with absolutely nothing to eat between her meals. She is allowed frequent drinks of water, but no crackers, cake, pretzels etc. But her meals are large, very large compared to those advocated by most of the text-books, as any of you will find, if you take the trouble to look up this subject. It has been the common practice to keep infants on a liquid diet until they reach eighteen months or later, beginning semisolid and solid food very gradually, even at that age.

Even at twelve months of age she was allowed only three meals, and these were fairly large, too: breakfast consisted of 1

tablespoon of strained oatmeal with milk, but without sugar, one slice of toast or zwieback, the juice of one orange, and one cup of milk. At dinner she had one-half, later one, baked potato or soft-boiled egg, two slices of toast, two tablespoons stewed prunes, and one cup of milk. At supper she had two slices of toast with butter, 2 tablespoons of prunes or apple sauce, and one cup of milk. From time to time the stewed fruit was changed in variety or orange-juice was given frequently in place of the stewed fruit, and whenever she seemed unwilling to eat much, she was given more milk in place of the solid food. As she grew in size and weight she was offered more variety and greater quantities of food, until she now takes for breakfast 3 or 4 tablespoonfuls of some well-cooked cereal, unstrained, 4 tablespoonfuls of stewed fruit or the juice of one orange, two slices of zwieback with butter, a soft-boiled egg, and one-half to one cup of milk. Her dinner now consists of about  $\frac{1}{2}$  ounce of beef, chicken, lamb, or boiled fish on alternate days, with a baked potato or 2 tablespoonfuls of boiled rice, 2 tablespoonfuls of spinach, two slices of toast and butter, and 2 tablespoonfuls of stewed fruit, with one-half to one cup of milk; on the alternating days on which she does not have meat she takes 4 tablespoonfuls of some cornstarch or custard instead. Her supper consists of three slices of toast and butter, 4 tablespoonfuls of stewed fruit, and one cup of milk, besides one of the following: a soft-boiled egg, a baked potato or cornstarch or custard, when she has not had one of these foods already in the day.

You can see how well she is; her mother will answer any questions about her. She has one or two bowel movements daily, usually two; from time to time the "soap-stick" is used to start her bowel movement. She is active, but still takes a short nap in the early afternoon. She goes to bed at 7 o'clock and sleeps until about 7 the next morning. There is no question about her intelligence; you can see that she is not nervous.

It was to speak of the rather radical changes in our method of feeding infants after weaning that I showed you this child. Last year I brought this matter before the Philadelphia Pediatric Society (at the meeting held January 9, 1917), but that discussion

was never published. We noticed, here, that many babies needed weaning between nine and twelve months, after having been fed upon undiluted milk without gaining. We noted, too, that many of these infants had several teeth, and investigation showed that they readily learned to take and apparently to assimilate semisolid and solid food. We found that milk was commonly either continued as the infant's sole food or was given in excessive quantities late into or even throughout the baby's second year. At this time beef-juice, beef, mutton, and chicken broths are also given in what appears to us to be greatly excessive quantities. Upon searching through the literature we found that most of the text-books on diseases of children advise from five to six meals a day, consisting chiefly of milk and broths. Crackers were advised in many instances also. Our experience here, covering, as it does now, eighteen years, has shown us that most, if not all, crackers produce much indigestion, whether eaten between meals, taken at meals, or given soaked in water or milk.

While it is important that the transition from a diet consisting solely of milk to one containing a variety of food-stuffs be made gradually, we firmly believe that the attempt to teach infants to take solid food should be made just as soon as the baby fails to gain upon milk alone and has at least four opposing teeth. The development of the baby, that is, his anatomic age, is a far better guide to feeding than is his actual age.

As a rule, every baby starts upon three meals a day at some time between ten and fourteen months; until he has learned to take at each meal enough of the variety of foods offered he is allowed a drink of milk, from a cup or spoon (thus abolishing bottles early), halfway between meals, but this is discontinued just as soon as the baby learns to eat three good meals.

The bad habit of an early bottle or the practice of giving orange-juice early in the morning is stopped; and breakfast is prepared as early as suits the mother. At first breakfast consists of a well-cooked cereal, preferably oatmeal, strained, with salt and milk added, but without sugar or cream (but farina, barley, rice, or other cooked cereal may be used for variety); a little zwieback, toast, or stale bread, and milk last, from a cup

or spoon. Much of the toast will drop out of the baby's mouth at first; but a few days' experience results in attempts at chewing and swallowing. Stewed fruit, apple sauce or baked apple, or orange juice are allowed, and more toast, zwieback, or stale bread; soon with butter added. The amounts are then increased to a saucer of cereal, three or four slices of toast with butter; a saucer of apple sauce or stewed fruit; and a soft-boiled egg is soon added.

We have noticed, as Kerley has,<sup>1</sup> that in some infants milk and orange-juice given at the same meal disagree; and that eggs do not agree with some babies early, as Grulee<sup>2</sup> pointed out. But we also found that most babies could take both milk and orange-juice together, and eggs soon after twelve months of age, without any symptoms developing.

Dinner consists at first of baked potato or a soft-boiled egg, with toast, zwieback or stale bread and butter, stewed fruit and milk. When the baby takes his egg for breakfast, it is omitted at dinner; but by this time a well-cooked green vegetable, well mashed or strained (spinach, peas, string beans, lima beans, asparagus, at first; later, cauliflower, Brussels sprouts, or boiled cabbage), has been added daily. Next chicken, beef or lamb, or boiled fish, finely chopped up, is given twice a week. Cornstarch, custard, or milk pudding are given on the days on which the baby gets no meat. No scraped raw beef is ever permitted; broths are allowed very seldom, not more than once a week, and then not more than 3 or 4 ounces; and beef-juice is only given in small quantities to the greatly undernourished children, as a stimulant rather than a food.

Supper consists first of toast, zwieback or stale bread, with butter, stewed fruit, and milk. Later a soft-boiled egg may be given (the second or even the third egg in a day, when eggs agree and the baby likes them); or a baked potato, cornstarch, custard, or milk pudding for variety. It is not fair to the baby to offer him exactly the same meals day after day. Variety will help much. The different cereals, always thoroughly cooked, may

<sup>1</sup> C. G. Kerley, *Practice of Pediatrics*, 1914, p. 97.

<sup>2</sup> C. G. Grulee, *Infant Feeding*, 1912, p. 143.

be given for breakfast, although oatmeal is undoubtedly best. When meat is given for dinner, no dessert is needed; but when the baby gets no meat, the cornstarch, custard, or milk pudding is allowed. Boiled rice may replace the potato at any meal, when the infant grows tired of potatoes. Enough should be given at each meal, but especially at supper; for the infant who eats a good supper will rest far better than one who only plays with his food and eats but little before going to bed.

Nothing to eat is allowed between meals, but drinks of water are encouraged. Especial condemnation must be expressed against the common practice of giving crackers between meals. Investigation among those babies who so often awake screaming an hour or two after going to sleep showed that they had very frequently, if not invariably, been fed crackers some time during the afternoon. Simply eliminating the crackers at once cured this condition in a large number of cases. Infants should take neither tea nor coffee; should have no alcohol, candy, fresh cake or bread; pickles, spices or condiments, including salad dressing; pork, veal, salt meat or salt fish; nothing fried; no nuts, no uncooked vegetables or fruit excepting the fruit juices; and no hard-boiled eggs.

It is important not only that the baby's food be properly prepared but also that it be given to the infant slowly, by some person endowed with patience and the ability to distract the baby's attention, amusing him by talking or story-telling, so that he unconsciously continues to open his mouth each time the spoon approaches his lips. In this way the baby will take sufficient nourishment at each feeding, reducing the action of his digestive functions to three times a day, with a normal period of rest between, instead of the old fashion of five or six meals daily, without sufficient rest between meals.

It is important, also, that the baby be fed regularly, having breakfast between 7 and 8 A. M.; dinner between 12 and 1 P. M., and supper between 5 and 6 P. M., so that he is in bed before 7 o'clock at night. It is also important to teach these infants to chew their food well before they reach the age of two years; and their teeth will need care by this time.

We have had excellent results in over 300 of our dispensary patients fed in this manner, besides many others in private practice. We believe that there is much less indigestion among infants fed in this way; possibly because these babies have gradually become accustomed to digest a variety of food-stuffs, and an occasional indiscretion does not produce the upset which so frequently results in the infant fed upon milk alone. An increase in growth, both in length and in weight, is noticed at once, and these babies, being better satisfied, rest better and appear to be much more contented than the many infants who are still fed upon milk and broths alone, even though they are over fifteen months of age.

The smallest of the 308 babies of whom I have full notes, covering the period from twelve to twenty-four months, weighed 10 pounds at twelve months and  $17\frac{1}{2}$  pounds at twenty-four months. The largest baby weighed 24 pounds at one year and 39 pounds at two years. Eleven of these infants out of 308,  $3\frac{1}{2}$  per cent., died from various causes during their second year of life. Some of the other babies showed the following gains (lowest,  $6\frac{1}{2}$  pounds; greatest, 15 pounds):

12 to 19 pounds;	$13\frac{1}{2}$ to 20 pounds;	$14\frac{1}{2}$ to $21\frac{1}{2}$ pounds;
15 to 23 "	$15\frac{1}{2}$ to 24 "	$16\frac{1}{2}$ to 23 "
$17\frac{1}{2}$ to 25 "	$18\frac{1}{2}$ to 29 "	19 to 27 "
20 to 33 "	(the child shown);	20 to $27\frac{1}{2}$ "
21 to 34 "	22 to 30 pounds;	23 to 31 "
24 to 35 "	24 to 39 "	27 to 35 "

We believe that a baby of twelve months, weighing about 20 pounds, deserves more than most text-books allow. Instead of from 800 to 900 calories a day, this infant needs from 1100 to 1200 calories. In other words, the infant of one year should receive from 30 to 35 grams of protein, from 35 to 40 grams of fat, and from 160 to 180 grams of carbohydrate daily.

At the age of two years, weighing 30 to 35 pounds, the baby should have from 1700 to 1800 calories a day, and not 1200 to 1500 calories. That is, he deserves from 35 to 45 grams of protein, from 70 to 75 grams of fat, and from 180 to 200 grams of carbohydrate daily.

The exact amount taken by each baby will vary at each meal

naturally. If there is something he likes, he will take all you give him of that, and less of other things. Then his appetite will vary, too, so that he may slight one or more meals a day. But he will usually make up on the next meal. Everyone who has frequently watched children eat has observed that they may refuse to eat one thing, such as spinach or soft-boiled egg, for instance, day after day, week after week. Oddly enough, some day the baby will apparently prefer the taste of this hitherto despised food-stuff and will eat all of his egg or spinach and decline all or many of the various other things making up the meal, things which he had been eating and enjoying up to that time.

Finally, in two tables I have worked out approximately the calories to be found in the ordinary meals offered to these infants when they are twelve months old and when they reach the age of twenty-four months:

## FIRST DIET LIST—BABIES OVER TWELVE MONTHS

<i>Breakfast:</i>	Grams.	Proteins, grams.	Fats, grams.	Carbohydrates, grams.	Calories.
1 tablespoon strained oatmeal	50	1.40	.25	6.75	32
1 cup whole milk.....	220	7.26	8.80	11.00	157
1 slice zwieback.....	15	1.47	.49	11.03	65
		10.13	9.54	28.78	254
<i>Dinner:</i>					
2 slices toast or stale bread...	40	4.60	.64	24.48	124
$\frac{1}{2}$ butter ball.....	8	.08	6.38	..	60
2 tablespoons stewed fruit...	83	.17	.67	31.00	134
1 cup whole milk.....	220	7.26	8.80	11.00	157
1 baked potato.....	130	3.77	.20	32.07	149
Or $\frac{1}{2}$ soft-boiled egg.....	25	3.30	3.00	..	42
Or 1 tablespoon boiled rice..	50	1.42	.05	12.20	56
		15.88	16.69	98.55	624
		or 15.41	or 19.49	or 66.48	or 517
		or 13.53	or 16.54	or 78.68	or 531
<i>Supper:</i>					
1 slice zwieback.....	15	1.47	.49	11.03	65
2 tablespoons stewed fruit...	83	.17	.67	31.00	134
1 cup whole milk.....	220	7.26	8.80	11.00	157
		8.90	9.96	53.03	356
Totals.....		34.91	36.23	180.36	1234
		or 34.44	or 39.03	or 148.29	or 1127
		or 32.56	or 36.08	or 160.49	or 1141

## LAST DIET LIST—BABIES OF TWENTY-FOUR MONTHS

<i>Breakfast:</i>	Grams.	Proteins, grams.	Fats, grams.	Carbohydrates, grams.	Calories.
2 tablespoons oatmeal.....	100	2.80	.50	11.50	63
$\frac{1}{2}$ tablespoon cream.....	10	.37	2.57	.36	27
2 tablespoons stewed fruit...	83	.17	.67	31.00	134
2 slices zwieback.....	30	2.94	.98	22.06	130
1 butter ball.....	15	.15	12.76	..	120
1 soft-boiled egg.....	50	6.60	6.00	..	84
$\frac{1}{2}$ cup milk.....	110	3.63	4.40	5.50	79
		16.66	27.88	70.42	637
<i>Dinner:</i>					
1 tablespoon spinach.....	50	1.05	2.05	1.30	29
2 tablespoons stewed fruit...	83	.17	.67	31.00	134
2 slices toast or stale bread..	40	4.60	.64	24.48	124
1 butter ball.....	15	.15	12.76	..	120
$\frac{1}{2}$ cup milk.....	110	3.63	4.40	5.50	79
$\frac{1}{2}$ baked potato.....	65	1.89	.10	16.04	75
Or 1 tablespoon boiled rice..	50	1.42	.05	12.20	56
1 tablespoon custard.....	15	1.10	1.71	3.03	33
Or $\frac{1}{2}$ slice lamb (beef or chicken).....	38	7.39	4.76	..	75
		12.12	22.28	74.48	575
		to 18.88	to 25.38	to 81.35	to 636
<i>Supper:</i>					
2 slices zwieback.....	30	2.94	.98	22.06	130
1 butter ball.....	15	.15	12.76	..	120
1 baked apple.....	120	.61	.58	29.30	128
$\frac{1}{2}$ cup milk.....	110	3.63	4.40	5.50	79
1 tablespoon custard.....	15	1.10	1.71	3.03	33
Or 1 tablespoon boiled rice..	50	1.42	.05	12.20	56
Or $\frac{1}{2}$ baked potato.....	65	1.89	.10	16.04	75
Or 1 soft-boiled egg.....	50	6.60	6.00	..	84
		8.43	20.45	56.86	490
		to 13.93	to 24.72	to 59.89	to 541
Totals.....		27.21	70.59	201.76	1702
		to 49.47	to 77.98	to 211.66	to 1814

## CLINIC OF DR. CHARLES S. POTTS

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### CEREBRAL PALSIES OF CHILDREN

WHAT are usually known as the cerebral palsies of children are those due to abnormal birth and those which occur as sequelæ of the infectious diseases. There may be other causes, as hydrocephalus, brain tumor, multiple sclerosis, and the abiotrophies, by this term meaning the premature death or degeneration of nerve-fibers due to inherent constitutional weakness. In this lecture we shall consider in detail only those commonly included under this title.

The largest group of such cases is that due to abnormal birth, often termed Little's disease, after the English physician who first called attention to it. This group comprises cases due to injury during birth from the use of the forceps or from prolonged or precipitate labor, to premature birth, and possibly cerebral disease in the fetus during intra-uterine life. Jaundice in the newborn is also a cause.

The lesion is most commonly located in the motor region, on one side or on both. The symptoms most frequently seen are, therefore, motor in character. The paralysis may be monoplegic, hemiplegic, paraplegic, or diplegic in type, according to the extent of the damage. Other symptoms of destruction of the upper motor neuron are present, as increased tendon-jerks, spasticity, and contractures. Symptoms also frequently observed are various forms of spasmodic phenomena, as the slow, vermicular movements, especially of the hands and sometimes of the feet, and facial muscles, known as athetoid movements or athetosis. In other cases choreiform or ataxic movements may be observed.

Paralyses of cranial nerves and speech disturbances occur more rarely. In some instances very little loss of power is present and the disability is due to the rigidity or spasticity of the mus-

cles. The very serious features of these cases is that many of them develop epilepsy and that many are mentally retarded. Thus, Spratling attributes 11 per cent. of 1070 cases of epilepsy to this cause. In an analysis of 5430 cases of mental defectives varying in severity made by different observers, approximately 12 per cent. was probably due to injury at birth. These figures represent cases so caused, and not those resulting from infectious disease acquired after birth, which cases will be considered later.

It must be borne in mind, however, that all do not develop these conditions and that some may be quite intelligent. Many of these cases are not recognized until some time after birth, frequently nothing is noticed until the child is old enough to sit up. When the condition is due to injury at birth, symptoms are present, as a rule, very shortly after, but to appreciate their nature the character of the lesions must be understood. This is hemorrhage either subdural or intermeningeal, due to tearing of the cerebral veins. Thrombosis of the superior longitudinal sinus may also occur. The hemorrhage with resulting clot may be unilateral, when hemiplegia usually results; or, it may be bilateral, when the effect is limited to the upper part of the brain and paraplegia follows, and if extensive, diplegia occurs. Hemorrhage is probably favored by the overlapping of the parietal bones during the molding of the fetal head. Owing to obstruction of the cortical venous system more or less softening of the cortex is liable also to occur. Rarely the hemorrhage may be arterial. More rarely the veins leading to the lateral sinuses may be torn as the occipital bone is pressed under the parietal, in which case the hemorrhage is subtentorial. Paralysis of some of the cranial nerves may be so caused. Many of these cases are either stillborn or die immediately after birth. Those who survive develop some of the following symptoms: if supratentorial, therefore involving the cortex, there is usually asphyxia after birth followed by restlessness, refusal to nurse, and convulsions, which may be unilateral. Examination will show some of the following conditions: Edematous or bulging anterior fontanel, muscular rigidity, subconjunctival hemorrhage, edema of the eyelids, inequality of the pupils, slow action of the heart,

and more or less papilledema. If the hemorrhage is infratentorial the early asphyxia is not so marked; in a few hours breathing becomes irregular and rapid owing to involvement of the nerves at the base of the brain, distention of the fontanel is not marked until late. There will be also marked rigidity of the muscles, especially those of the neck, sometimes with retraction of the head. The presence of these symptoms indicates that a lumbar puncture should be done, when the fluid will usually be bloody and under increased pressure.

The amount of blood is apt to be greater in infratentorial than in supratentorial hemorrhage. These symptoms frequently do not attract attention, and, as has been said, nothing is noticed until the child is old enough to sit up. Then the parents will observe that it is unable to do so. Even when the child is sat up the head will fall forward or roll about, owing to weakness of the muscles of the neck. It may be noted that the child does not move its limbs about as it should or that it moves those of one side and not the other. In some cases convulsions may occur at intervals, or various spasmodic or choreiform movements of the limbs are noticed. These may be mistaken for chorea. If examined, more or less rigidity of the limbs will be found, according to the type of the paralysis previously mentioned.

J. D., *æt.* twelve weeks, is an example of a probable birth injury with resulting hemiplegia. This child had to be delivered by version. On the third day it had a series of convulsions lasting for twenty-four hours. At the present time the child is paralyzed on the right side, and it will be noticed that the defective limbs are rigid and that the knee-jerk is possibly somewhat increased. The Babinski reflex is also present, but in a patient of this age this has no pathologic significance, as the phenomenon is normal in infants up to three or four years of age.

H. B., forty-two years of age, has a history which is very characteristic of the condition of which we are speaking. He is the second child of fifteen. Of this number, one was still-born; one was prematurely born at eight months and lived five days; another had convulsions during the first week and died. The others are living and, with the exception of the

patient, are healthy. The birth of this patient was normal; no instruments were used; labor was said to be easy. The mother, however, during the ninth month of her pregnancy fell down a flight of steps, but noticed no ill effects. On the third day after birth the child had convulsions. From this time nothing was noticed until the time when he should have been sitting up, when it was found that he was unable to do so. He did not walk until he was nine years of age. The present condition is one of marked rigidity of all four limbs. The patient is able to walk, but with great difficulty, and with dragging of the feet along the ground. The knee-jerk, biceps- and triceps-jerks are increased and an ankle-clonus is present. There is marked speech disturbance, so that it is difficult to understand the patient. What especially attracts attention, however, are the continuous, slow movements of extension and flexion of the fingers and hands, also at times of the arms, and the irregular contractions of the facial muscles when he attempts to speak. These movements are those known as athetoid movements. The patient's mental condition is deficient, although he understands fairly well what is said to him.

These patients represent types of the usual conditions following birth injury. However, other symptoms may occur, but not commonly. Thus, cases have been described in which instead of spasticity there is extreme flaccidity of the limbs. In addition, the child for several years is unable to walk or stand, and if it does become able to do so, ataxia will be noted. Disturbance of articulation and mental deficiency are also usually present. There is no muscular atrophy. The knee-jerks may be either absent, normal, or increased, and the Babinski reflex may or may not be present. This type has been termed either the "atonic astasic type of infantile cerebral paralysis" or "cerebral diplegia of flaccid atonic type." In the few cases that have come to autopsy lesions in the frontal lobes were observed.

A case somewhat resembling this type is W. B., æt. three years. This patient is a first child and an instrumental delivery was necessary. Three days following birth he had a series of convulsions lasting for several days. He began to walk at the

age of sixteen months. His gait, however, was somewhat unsteady. Examination will show an unsteady gait; the hands are used clumsily when he attempts to touch or pick up objects. There is also some slight tremor of the hands. The knee-jerks are present, but not increased, and no undue rigidity or spasticity will be noticed when the limbs are moved. The patient since birth has had from time to time convulsions of an epileptiform nature. His mental powers are not what they should be at his age. Speech is somewhat indistinct.

In some instances ataxia of a cerebellar type develops. In these cases there will be the usual history of difficult labor, backwardness of mental and physical development, and when the child learns to walk the gait will be noticed to be very unsteady, resembling that of a drunken man. Ataxia of the hands may also be present. Defective speech, which is slow and jerky, nystagmus, and tremor of the head may sometimes be observed. In these cases muscular power is good and there is frequently a considerable degree of improvement. Such cases have been termed the cerebellar type; the lesions are supposed to involve the cerebellum.

Hunt, of New York, has recently reported several cases which he has termed the "ataxic type of cerebral birth palsies," and, on theoretic grounds, believes that the symptoms are due to the hemorrhage and resulting lesions being limited to the parietal lobes. The ataxia is bilateral, involving the arms and legs, and is present when the patient is lying down. Speech is defective. The cases which Hunt has described resembled symptomatically very much those designated as the cerebellar type. There has been no actual demonstration of the lesions in either of these classes of cases.

As has been stated, there are certain cases in which the symptoms described are not due to injury, but occur in children born prematurely. They are due to a failure of the motor centers and of the pyramidal tracts to develop. In some the only abnormality is a fineness and diminution in number of the fibers of the pyramidal tracts. The symptoms, except that those described as occurring just after birth do not occur, are

similar to those following injury with hemorrhage. Mental deficiency and epilepsy do not develop quite so frequently as in those due to hemorrhage.

M. S., two and one-half years of age, is an example of this class. She was a six months' child and weighed one and one-half pounds at birth. At the present time she is unable to stand or walk, but can sit without assistance. Both arms and legs are excessively spastic. Contractures will be noticed; the kneejerks are markedly plus.

Another example of this type is F. H., twenty-eight years of age. He was a seven months' child and did not walk until he was four years of age. There is extreme spasticity of the muscles of both arms and legs; the tendon reflexes are all increased. The Babinski reflex is also present. It will be noticed that on testing the muscular strength of this patient it is very good, and he is an example of that type in which the disability is due more to excessive rigidity of the muscles than to loss of strength. He has, as will be seen, very marked athetoid movements of the arms and contortions of the facial muscles. In this case the movements of the limbs are so violent that the hands will fly up, at times striking him in the face.

Some of these cases may be due to hereditary syphilis, and in the patient, H. B., in whose family history we find that three other children probably had the same condition as the patient, the possibility of hereditary syphilis seems to be strong. Other cases may possibly be due to infectious disease in the mother during pregnancy.

A not uncommon cause of paralysis in children is apoplexy from hemorrhage, thrombus, or embolism following one of the acute infectious diseases of children. In such cases the child, of course, is normal until it acquires either diphtheria, measles, whooping-cough, or similar disorder. The usual history is that either while convalescing or during the height of the disease convulsions occur, which may be limited to the affected side. Consciousness is lost for a time, and when regained the patient is found to be hemiplegic, and possibly, if the lesion is on the left side, also aphasic. The resulting condition is similar to hemi-

plegia following apoplexy in the adult, and frequently, which is not the case in the adult, epilepsy and imbecility result in addition.

It does not seem to be generally known that while one is most liable to apoplexy between the ages of forty and sixty years, exclusive of this period he is more liable to it during the first ten years of life than at any other time. Acutely developing paralysis following infectious disease is not always due to apoplexy, but may be due to inflammation of the cortical cells, or encephalitis. In addition, the cells composing the cranial nerve nuclei may be affected and also the cerebellum. In the former case there will be cranial nerve palsies; in the latter, ataxia in addition to paralysis of the limbs.

The distinction, at the time of onset, between apoplexy and encephalitis may be difficult. In the latter the onset may not be so sudden; there may be no loss of consciousness; headache and some rigidity of the muscles of the neck may be present.

An example of paralysis due to cerebral lesion following infectious disease is M. S., nineteen years of age. This patient was born after a difficult labor, but was normal until he was two years old. At that time he contracted what was said to be diphtheria. During convalescence from this disease he was seized with a series of convulsions lasting seven hours. When he regained consciousness he was found to be paralyzed on the left side. Ever since this time at varying intervals he has had convulsions of epileptiform type. At the present time his gait seems to be of the hemiplegic type. There are some contractures of the muscles of the left arm and inability to use the arm. The mental condition of the patient is fair and he has been able to make a living by selling newspapers.

If the brains of these patients, in which injury was due to birth accident or infection, are examined some years after birth the following may be found: Atrophy and sclerosis, either of a group of convolutions, an entire lobe, or of a hemisphere; por-encephalus, meaning a cavity in the surface of the brain and cysts which may extend into the ventricles.

The diagnosis of meningeal hemorrhage at birth is very important, for, as we shall tell you later, something may be done

then to lessen the probability of permanent disability. Cyanosis of the head with convulsions, followed by coma, especially when subsequent to a difficult labor, is very significant. At the same time it must not be forgotten that this accident may also occur during easy labor.

The occurrence of these symptoms should lead to careful examining of the patient for any of the symptoms previously mentioned as being present immediately after birth. It must be borne in mind, however, that asphyxia after birth may be due to disturbance of either the fetal circulation, anemia from rupture of the cord, and possibly tetany and hydrocephalus. Later in life cerebral birth palsies may have to be distinguished from injury to the brachial plexus occurring during labor, the so-called *obstetric* paralysis or birth palsy of Duchenne. In this the disability is confined to one arm. If examined early there may be observed tenderness and swelling in the neck. Later the arm will be flaccid; the biceps-jerk and possibly the triceps will be absent, and the muscles will not respond to the faradic current as in cerebral palsy. The greatest weakness will be found in the muscles about the shoulder. In the cerebral palsies this is usually in the muscles of the arm and hand.

The athetoid and choreiform movements which have been described have been mistaken for Sydenham's chorea. It must be borne in mind that in not a few of these cases there may have occurred marked improvement in power and spasticity, and in such, if the physician is not extremely careful, the mistake will be made. Some evidence, such as the Babinski reflex, of damage to the pyramidal tracts will usually be found, and the history of the patient should make the case clear. In some cases of chorea there *may* be motor weakness, but this develops sometimes after birth, and when present the limbs are not spastic.

The atonic-astasic cases may be confounded with either myotonia congenita or amaurotic family idiocy. Both of these conditions appear, however, after the child is several months old. In the former the tendon-jerks are absent, and if the patient should be more than two years of age the Babinski reflex will be absent. In the latter condition there will be failure of

vision progressing to blindness, with the peculiar appearance of the optic nerve which is present in this disease. It is also a family disease most often occurring in Jews.

There is a possibility that acute anterior poliomyelitis may be mistaken for this condition, especially as cases have been reported in which the disease apparently occurred *in utero*. In these, however, the paralysis may involve only a group or groups of muscles. It is not apt to be hemiplegic or diplegic in distribution, and the affected muscles will be atrophied and flaccid and show a more or less developed reaction of degeneration. The tendon reflexes in the affected limbs will be diminished or absent.

The ataxic type may be mistaken for hereditary ataxia, either of the Friedreich or cerebellar type. In the former there will be nystagmus; the knee-jerks are absent; hyperextension of the toe is usually present. The disease develops at about fourteen to fifteen years of age, and has been frequently present in different generations of the family. In the latter optic atrophy will be present. Multiple sclerosis occurring in early life may present somewhat similar symptoms to the spastic type, but its development several years after birth is sufficient to exclude it. Hydrocephalus also may cause paralysis, either hemiplegic or diplegic. The development of the peculiar shape of the head, however, is sufficient to distinguish this condition.

A number of different forms of spastic paralysis, some of which are due to cerebral lesions and some to spinal, have been described, which in their symptoms resemble somewhat the spastic type of cerebral birth palsies. They are generally family diseases and may be either diplegic or paraplegic. They may develop in the patient at any period between the ages of several months and eighteen to twenty years. Mental deterioration is frequent, and optic atrophy, leading to blindness, nystagmus, paralysis of cranial nerves, ataxia, speech defects, and tonic or clonic spasm are symptoms. Some of these cases are due to a diffuse sclerosis of the cortex; others, to degeneration of the pyramidal tracts; in other words, are abiotrophies. The differential diagnosis between the spinal and cerebral types

may be impossible to make during life. Children attacked with these disorders are normal during the first few months or years. Some of the conditions may be due to hereditary syphilis. In many respects they resemble multiple sclerosis. The term "aplasia axialis extracorticalis congenita," or Pelizaeus-Merzbacher's disease, has been applied to a group of such cases in which a diffuse sclerosis is found after death. Krabbe has described a group somewhat similar to amaurotic family idiocy, or Tay-Sachs disease, which also belongs among these cases. This latter disease may develop some years after infancy.

Spina bifida occulta may cause paralysis in children.  $x$ -Ray examination will reveal its presence. Progressive lenticular degeneration or Wilson's disease causes spasticity of the limbs, speech defects, tremor, etc., without loss of muscle power. It does not develop until the child is several years old, sometimes seventeen or eighteen.

Cerebral paralysis following infectious disease may be mistaken for poliomyelitis. The history of onset is different. The muscles are firm and respond normally to the faradic current. The tendon-jerks are increased. The Babinski reflex is present. None of these symptoms are present in poliomyelitis. The fact that in the cerebral cases the affected limbs sometimes remain smaller than those of the normal side may lead to error, but attention to the occurrence of the symptoms just enumerated should make the case clear.

Multiple neuritis may also be the cause of paralysis following an infectious disease. In these cases, however, it is of the flaccid type. There is muscular atrophy and absent tendon reflexes, and, with the exception of the diphtheritic form, there will be tenderness over the affected nerves. In cases following diphtheria, tenderness and pain are usually absent.

Regarding the prognosis it may be said that if the child does not die during early life there is often a tendency to improvement, so much so that the paralysis may largely disappear. This is not the rule, and more or less disability is found in most cases. If epilepsy and mental enfeeblement occur, of course they are not recovered from, but they may be ameliorated by proper treatment.

The question of prophylaxis is of great importance in considering the group due to injury at birth. Statistics show that more cases result from prolonged labor than from the use of the forceps. Sachs, in his book on the Nervous Diseases of Children, writes that tedious labor is much more dangerous than the forceps, and that most cases occur in first children. Sharpe and Farrell, in their recent paper, say that prolonged labor should be avoided whenever possible, that the cesarean operation is less dangerous to mother and child than the high forceps. They also lay stress on the importance of carefully watching every child for symptoms and signs which have been mentioned as occurring early. In those cases in which the hemorrhage involves the convexity, early operation holds forth some hope of relief. Cushing in 1905 reported 4 cases of this kind operated on shortly after birth. He opened the skull and removed the clots. He got good results in 2. This has since been done by others with more or less success. As most of these patients either die soon after birth or are doomed to a life of disability and suffering, such operation seems to be justified when the diagnosis of hemorrhage can be made. It is true that some do not develop epilepsy and do not become mentally impaired. The motor disability may be slight, and in the hemiplegic cases especially the patient may be self-supporting. These instances, however, are very much in the minority. Lumbar puncture, which has been previously mentioned as useful for diagnostic purposes, may also be of considerable therapeutic value at this period. If evidences of distention should appear after puncture has been made, it should be repeated. Some excellent results have been obtained by this procedure.

Bearing in mind also the possibility of hereditary syphilis, Wassermann tests of the parents should be made, and if positive in either, proper treatment should, of course, be instituted. The majority of the cases which survive are due to involvement of the convexity, and Sharpe and Farrell, of New York, have operated on a number of selected cases of this type some time after birth. They consider the indications for their operation to be signs of increased intracranial pressure, as shown by papilledema and increased pressure of the cerebrospinal fluid

when removed by lumbar puncture, with a history of difficult labor and convulsions and asphyxia after birth. They opened the skull over the motor area on one or both sides, as required. In most cases meningeal cysts were found and the brain cortex is apparently healthy. In other words, the interference with function was due to pressure. The cysts were punctured, and their walls removed. Of 65 cases so treated there was marked improvement in 25. The youngest child was two and one-half years of age and the oldest seventeen. Nine died after the operation; 8 within two years; 19 showed no change, and 4 disappeared from observation. The authors state that the sooner the operation is done, the better is the outlook for improvement. Following the operation various mechanical and orthopedic measures must be employed as indicated.

Such measures may also be of considerable service in cases in which operation has not been done. Deformities may be relieved by lengthening tendons, by tenotomies, massage, muscle education, and electricity. In using the latter it should always be applied to the muscles, which are being overcome by their antagonists. Thus in the case of the arm the extensors should be treated and not the flexors. If the patient has convulsions of the Jacksonian type, exploratory operation is indicated. This to give hope of success must be done early, as the longer the convulsions continue, the more probably will the brain contract what is termed the convulsive habit, in which event the convulsions may continue even if the source of the trouble is removed. Cases in which the spasticity is great, and motor weakness not marked, may be relieved by the operation of rhizotomy, by which we mean cutting some of the posterior spinal roots. In the case of the legs this will be those of the lumbosacral plexus, and of the arms, the brachial plexus. By this means an artificial tabes is caused, with resulting hypotonicity of the muscles, and, while some ataxia may result, there is often considerable relief to the previous spastic condition and gain in the function of the limbs. The epileptiform convulsions may be limited in frequency by the usual treatment for epilepsy. Otherwise there is no indication for drugs in these cases except symptomatically.

## CLINIC OF DR. LEON JONAS

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#### DIABETES

##### **Clinical Determinations of Blood Sugar and Acidosis as Guides in the Management of Diabetes**

THE subject of our talk will be diabetes mellitus. This is a large subject, therefore I shall only discuss part of it today, namely, the significance of blood sugar and certain phases of acidosis.

During the past few years methods have been discovered for the determination of blood sugar and for the study of acidosis that not only require small amounts of material for the examination, but come within the scope of laboratory work which an internist might do.

If a case of glycosuria presents itself, one must first determine whether it is due to actual diabetes mellitus, or is to be classed as renal glycosuria, or as a consequence of head injury, drugs, or other possible causes. The chief differential diagnosis is between diabetes mellitus and renal glycosuria. This is especially so since our present methods of investigating glycosuria have shown that cases of renal glycosuria are more frequent than we previously supposed. The prognosis and treatment being different for each condition, correct diagnosis is paramount.

The determination of the blood sugar and urine sugar will best aid us to make this differentiation. In renal glycosuria the blood sugar is always within normal limits, and the amount of glucose appearing in the urine is not affected by increasing the carbohydrate intake. In diabetes mellitus when there is a glycosuria the blood sugar concentration is above normal, with

an exception or two, which will be discussed later. The blood sugar must reach a certain concentration before glucose appears in the urine, this concentration being practically the same in all normal individuals. Technically, this is called the threshold, a term indicating that concentration of glucose above the specified degree that will lead to excretion of sugar in the urine.

The limits of normal blood sugar, the influence of diet upon it, and the normal threshold must be understood before the proper interpretation of blood sugar values can be made.

A normal individual after starving at least six to eight hours will have a blood sugar concentration which will vary between 0.07 and 0.1 per cent. If the blood is examined one-half hour to two hours after eating a meal containing carbohydrates it will show values between 0.1 and 0.15 per cent., depending upon the amount of carbohydrate in the diet.

Hamman and Hirschmann<sup>1</sup> have shown that glucose does not appear in the urine until the blood sugar has reached a concentration of 0.17 per cent., which we will consider the normal threshold. In certain cases of diabetes we may have a lowering of the threshold, while cases complicated by nephritis may show an increased threshold. We will consider the blood sugar to be above normal, or that a hyperglycemia exists, when the concentration is above the normal threshold one to two hours after a meal containing carbohydrates, or above 0.11 per cent. six to eight hours after fasting.

Again returning to our case of glycosuria; if it is diabetes mellitus, what are the possible blood sugar findings, and when should we make the examination? Make an examination after the patient has fasted six to eight hours or, preferably, in the morning before any food has been taken. In mild cases we will find that the blood sugar may vary from the upper limit of normal to figures in excess of the normal threshold 0.17 per cent. If below the threshold value the urine at this time will probably be sugar free.

Having obtained the starvation level, it is advisable in some cases to determine the influence of carbohydrates on the level of blood sugar. This procedure is to be recommended if the

fasting level is below the normal threshold. In other words, test the carbohydrate tolerance. This is best done by examining the blood one-half hour to two hours after taking varying amounts of carbohydrate food or weighed quantities of glucose. Testing the carbohydrate tolerance is especially advisable in cases where the diagnosis is questionable. This will best be illustrated by the following 2 cases:

CASE I.—McC., a physician, while on duty at a naval base hospital in France developed symptoms of diabetes, including sugar in the urine. He was under treatment for diabetes in France, and recently returned to this country on sick leave. His basal blood sugar on the first examination at the University Hospital was 0.1 per cent. After taking 50 grams of glucose the blood sugar rose to 0.12 per cent. and no sugar in the urine. On the second examination, two weeks later, the starvation level was 0.1 per cent., and after taking a very heavy carbohydrate meal rose to 0.14 per cent. From the above results one would interpret that at the time of these examinations there was no apparent impairment of his carbohydrate metabolism. With the history of glycosuria, although there is no impairment at present, he should be observed regularly for some time.

CASE II.—F. L. B., a physician, was examined for life insurance and refused because of glycosuria. He frequently examined his urine thereafter and found it persistantly negative for glucose. It was then decided to determine his tolerance by giving 100 grams of glucose. After taking the glucose the blood sugar rose to 0.23 per cent. and sugar appeared in the urine. A normal individual after taking 100 grams of glucose should not have glucose in the urine nor have a blood sugar above 0.15 per cent. The patient, therefore, has a lowered carbohydrate tolerance. This is the counterpart to the so-called alimentary glycosuria. This patient and many similiar cases have an impaired carbohydrate tolerance, although the glucose only appears in the urine after taking very large amounts of carbohydrates. It would seem that they are cases of potential diabetes, particularly since we know that the continual overstraining of the carbohydrate metabolism will eventually lower the carbohydrate

tolerance, and possibly convert these cases from potential to active cases of diabetes. They need to be cautioned against partaking of large amounts of carbohydrates.

In the cases with a more limited carbohydrate tolerance and the severe cases we may find that the starvation level, particularly if the previous dietary restrictions have been lax, varies from just above the normal threshold to 0.5 per cent.

The vast majority of physicians in treating their cases of diabetes depend wholly upon the presence or absence of glucose in the urine as the gauge of the patient's tolerance. Since, however, better methods to determine that the patient is within the limits of tolerance are available, and at the same time can be done accurately by all physicians instead of a relatively limited few, it is important to discuss them. The Lewis and Benedict<sup>2</sup> method of determining blood sugar, or one of its modifications, such as Epstein,<sup>3</sup> meet these requirements. These methods require small quantities of blood, 0.5 to 2 c.c., depending on the method selected. The blood is obtained either by puncturing the finger or from a vein. The protein of the blood is removed by precipitating with picric acid solution and then either centrifuging or filtering. The clear filtrate is boiled with sodium carbonate solution, and the color thus produced is determined by a colorimeter, using a known standard solution of a pure glucose or picramic acid. In the Epstein method the colorimeter is patterned after the Sahli hemoglobinometer. The determination of blood sugar is of value as a guide in increasing the carbohydrate intake. This is especially so in the cases in which the tolerance is improving, and in the so-called cured cases.

After each increase in the carbohydrate intake the blood sugar should be determined one-half hour to two hours after eating to see if the added carbohydrate is within the patient's tolerance. This is best illustrated by the following case in which the carbohydrate in the diet has been increased from 70 to 200 grams without overtaxing the carbohydrate tolerance.

T. C., a boy nine years old, was admitted to the University Hospital in January, 1918, with glycosuria, a mildly positive ferric chlorid reaction, blood  $\text{CO}_2$  59 volumes per cent., and a

hyperglycemia of 0.29 per cent. (after taking food). He was very quickly made sugar free and showed a fasting blood sugar of 0.09 per cent. With each addition of carbohydrate to the diet his blood sugar was determined, in most instances one to two hours after breakfast, and on several occasions after his midday meal. At no time did it go above 0.14 per cent. The blood sugar has been determined one or more times each month since the boy left the hospital, with the results stated above, except that in November, 1918, the blood sugar rose to 0.23 per cent. after the midday meal and glycosuria was present. In December, 1918, it rose to 0.18 per cent. and a trace of glucose appeared in the urine. On investigating the cause of the hyperglycemia the parents stated that they were no longer adhering to the diet and were permitting the patient to buy candy, ice-cream, and other carbohydrate-rich food.

Dr. S., admitted to the University Hospital in 1918 with an acute severe diabetes. At the height of the disease he eliminated as much as 16 liters of urine, 40 grams of ketones in terms of acetone, and rapidly lost weight. After a month's treatment he was made sugar free and his carbohydrate tolerance rapidly improved. His blood sugar was frequently determined during his stay in the hospital and never showed a hyperglycemia after he became sugar free. He learned to examine his own blood, and frequent examinations showed a normal glycemia until August, 1918, when, after an attack of influenza, a hyperglycemia and glycosuria developed. His carbohydrate tolerance on leaving the hospital in 1914 was about 30 to 50 grams, and gradually improved until 200 grams were taken without overtaxing his tolerance. He was readmitted to the hospital December, 1918, with glycosuria and a fasting blood sugar of 0.26 per cent. The history showed that he was apparently well until August, 1918, when his tolerance became impaired. He has very easily been made sugar free on a diet very low in carbohydrates.

We occasionally meet old cases of mild diabetes in which nephritis has insidiously developed and, as a result of the nephritis, the glycosuria has become markedly decreased and at times disappears. If we depend on the presence or absence

of glycosuria as the gage of the condition of such a case, we might be deceived, because, as the nephritis advances, the permeability of the kidneys becomes less. With the permeability of the kidneys decreased it becomes more difficult for the kidneys to excrete glucose. A higher blood glucose concentration is necessary before glucose appears in urine, or, in other words, a higher threshold for glucose occurs.

Occasionally in severe cases the threshold becomes lower and glucose may appear in the urine after blood sugar concentration of 0.13 per cent. is reached. Again, there are some cases in which it is not advisable to reduce the blood sugar to the normal starvation figures. In these cases, if the fasting blood sugar level is brought by dieting within normal limits and then more carbohydrate added to the diet, the rise of blood sugar may be out of proportion to the amount of carbohydrate added. This is due, as suggested by Mosenthal<sup>4</sup> and his co-workers, to the endeavor on the part of the organism to adjust itself to the increase.

Let us now discuss some features of acidosis, which is an important complication of diabetes that requires laboratory tests for its recognition and proper valuation. A severe acidosis is present in all cases of diabetic coma, therefore its early detection and prompt treatment is very important to prevent coma. Acidosis in diabetes is due to the accumulation in the blood of the intermediary products of fat metabolism, namely, beta-oxybutyric acid, diacetic acid, and acetone, which collectively are called the ketone bodies. Normally these products do not appear in the urine, or in such small quantities that they cannot be detected by the available qualitative methods. In cases of diabetes with any degree of acidosis we are usually able to detect diacetic acid by the ferric chlorid reaction. Throughout this talk the ferric chlorid reaction will be considered the most generally available for the qualitative detection of ketonuria. The best methods, however, for detecting acidosis are those which measure the alveolar  $\text{CO}_2$  tension and the blood  $\text{CO}_2$  content. The Marriott<sup>5</sup> method of determining the alveolar  $\text{CO}_2$  is so devoid of the necessity of high technical skill that it can be accurately operated

by any physician. The Van Slyke<sup>6</sup> method for determining the blood  $\text{CO}_2$  requires some practice, but the technic of its operation is readily acquired. The Marriott method for determining alveolar  $\text{CO}_2$  tension can be conducted at the bedside. The Van Slyke blood  $\text{CO}_2$  method requires a special gas buret devised for this test, a barometer, a high-speed electric centrifuge, and other minor pieces of apparatus, which, together with the financial outlay, will restrict its use to hospitals and to a relatively few physicians with well-equipped private laboratories. Accepting 60 to 70 volumes per cent. as the normal blood  $\text{CO}_2$  and 40 as the normal alveolar  $\text{CO}_2$  tension, any reduction in these figures is evidence of acidosis. Needless to say any increase or decrease of the blood  $\text{CO}_2$  content or of alveolar  $\text{CO}_2$  tension will tell us whether the acidosis is being successfully combated.

Many physicians use sodium bicarbonate in the treatment of the acidosis, while others oppose it, particularly Joslin.<sup>7</sup> He objects to its use on the following grounds: that it sets free acid bodies which are combined and harmless in the body, and thus does harm; its use over a long period of time may deplete the body of its necessary body salts, particularly sodium chlorid; it causes nausea and vomiting. Further, he holds that large quantities of urine must be voided to remove the salts of the acid. This causes the ingestion of large quantities of liquids, overloading the stomach, and the excretion of much acid, which frequently overwhelms the kidneys, causing them to cease to act. The constant use of an alkali appears to promote the constant excretion of acid bodies in some cases. When the alkali is withheld the ketonuria disappears, only to reappear on the use of alkali. Sodium bicarbonate is unquestionably contraindicated in some cases, but, on the other hand, its use is most beneficial in certain cases. Theoretically, it would seem that the use of sodium bicarbonate is the logical treatment for the following reasons: bicarbonate of soda is the main buffer substance which neutralizes the foreign acids that accumulate in the blood. Therefore acidosis has been defined as any decrease in bicarbonate reserve in the blood. The administration

of bicarbonate of soda replenishes the depleted bicarbonate reserve.

When sodium bicarbonate is used in the treatment of acidosis the determination of the alveolar  $\text{CO}_2$  tension or the blood  $\text{CO}_2$ , preferably the latter, are the best indices to determine whether sufficient sodium bicarbonate has been given. It is known that excessive amounts of sodium bicarbonate do more harm than good. Formerly the presence of an alkaline urine was the signal that sufficient alkali had been given. It has, however, been demonstrated that in many cases of diabetes the kidneys are not normal, and that the urine in these cases does not become alkaline until the blood bicarbonate reserve is far in excess of normal. Cases have been reported in which sodium bicarbonate has been given in such quantities to create a state of alkalosis with tetany before the urine became alkaline. Therefore when sodium bicarbonate is used do not depend on the reaction of the urine, but on the blood  $\text{CO}_2$  content. If the blood  $\text{CO}_2$  is normal and the urine acid, disregard the latter. If the blood bicarbonate has been raised to normal and the urine still shows ketones, do not think that you have successfully combated the acidosis and that the danger of coma is past. Occasionally sufficient sodium bicarbonate will be given to raise the blood  $\text{CO}_2$  to normal, but the patient nevertheless may die in coma.

Physicians who do not use bicarbonate of soda in the treatment of diabetic acidosis would not experience this condition, but would find a low  $\text{CO}_2$  reading. A normal blood  $\text{CO}_2$  reading with a negative diacetic acid reaction is what we should strive for. In cases of acidosis it is advantageous to determine the alveolar  $\text{CO}_2$  tension or the blood  $\text{CO}_2$  content daily.

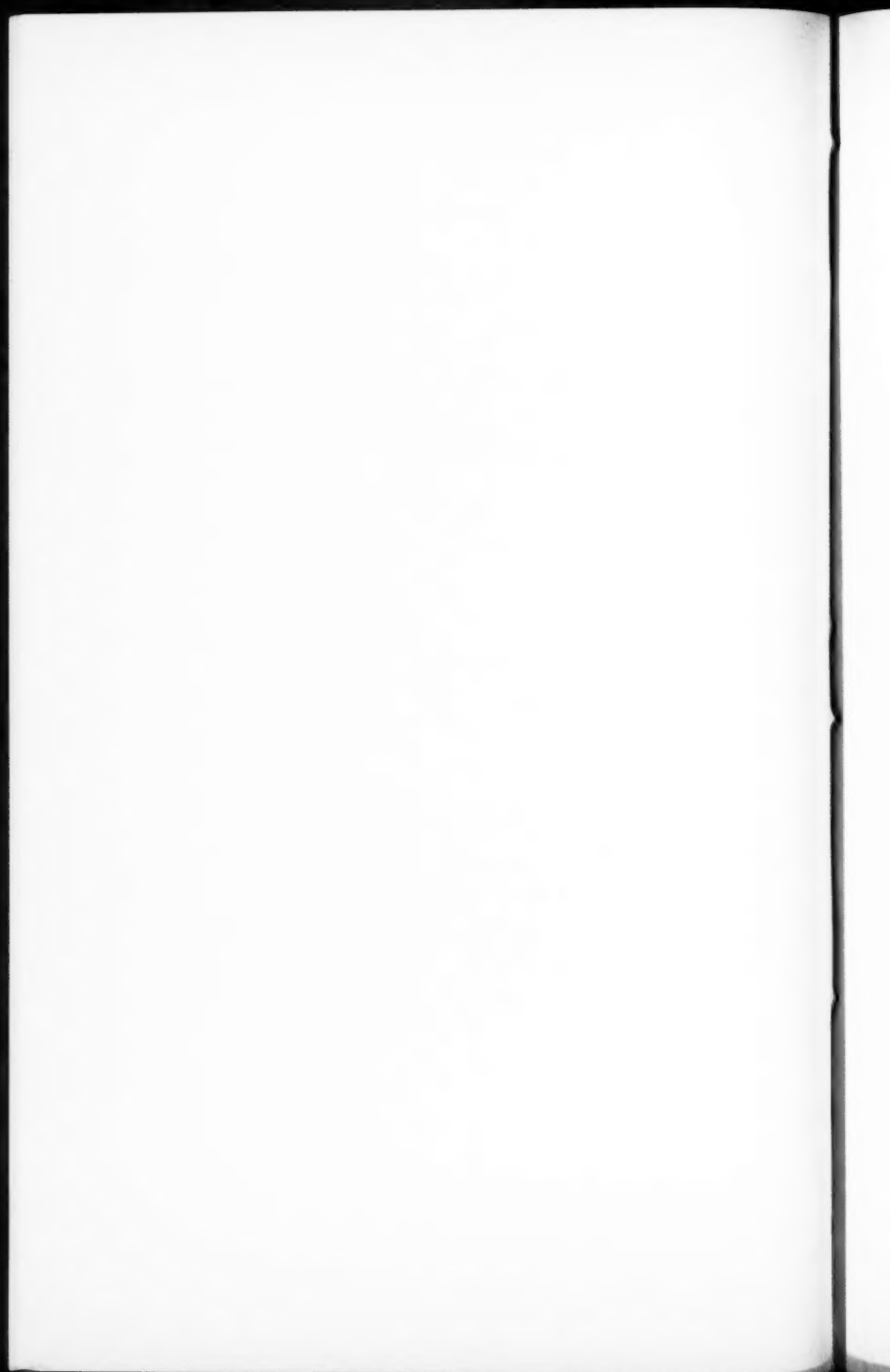
The object of this talk has been to outline the use of those tests which are within the reach of all clinicians. With the employment of tests for studying the blood sugar, urine sugar, blood  $\text{CO}_2$  content, or alveolar  $\text{CO}_2$  tension, and the use of the ferric chlorid reaction for the detection of diacetic acid the physician has in his possession the important methods for the proper treatment of diabetes along modern lines.

Of course, if he is able to make or obtain quantitative deter-

minations of the ketone bodies in both the blood and urine, the urinary ammonia nitrogen, the total nitrogen of the urine, and the blood lipoids, etc., it is a further advantage.

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## CLINIC OF DR. DAVID R. BOWEN

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### X-RAY DIAGNOSIS OF LUNG DISEASES

#### **A Presentation of Cases with Typical Findings, and of Others in which Clinical Findings are Proved Incorrect.**

WE have arrived at the point where it is quite proper to say that there is no intrathoracic condition in which the diagnosis may be considered complete if x-ray study has been omitted. In very many instances x-ray study will be found to have furnished the most important data for diagnosis, and in not a few cases it will be found that it has not furnished vitally important information, but that no part of this has been revealed by other clinical methods.

Why then has it not been more commonly employed? The answers are many, but all are included in one—lack of understanding.

Beginning with the scant attention given to roentgenology in medical colleges, we are further confronted by the fact that the majority of monographs and papers upon general topics in medicine are still written by men who have had but little training in, or experience with, roentgenology, and that there is still an exceedingly large percentage of patients whose medical care is entrusted to physicians who know but little of the possibilities of x-ray diagnosis. As prolonging this condition there has been an attitude of self-satisfaction on the part of the clinician and a lack of aggressiveness on the part of the roentgenologist.

Until recently, at least, one might be justified in classifying roentgenologists in two principal groups; the one absorbed in the scientific detail of the fascinating study and having little inclination and less time for propaganda; the other, a larger class,

those who for one cause or another have occupied inferior positions both as to hospital and private practice. Much valuable contribution from this class has had limited distribution.

All this now becomes a matter of history. At the various training camps, but particularly at Camp Greenleaf, the value of roentgenology has been so efficiently taught that we may confidently expect to see this method come into its own with a rapidity quite out of keeping with its, until now, rather slow recognition.

The complete study must be fluoroscopic as well as roentgenographic. It is our practice to examine every thorax patient fluoroscopically before making plates. Beginning at the apices we observe whether the aëration is equal on both sides both with ordinary and forced breathing. Then with the diaphragm set to illuminate a horizontal strip of screen from 1 to 2 inches wide we sweep slowly downward over the entire chest looking for unequal aëration. If such areas are found they are carefully studied with large and small diaphragms. Next the arch of the aorta is observed, both in anteroposterior and oblique positions, for dilatation or aneurysm. The auricles and ventricles of the heart are then studied. The left auricle and both ventricles are normally easy to distinguish, while the right auricle if hidden is easily observed in the semi-oblique view. No physical sign of pericardial effusion appears so early as does the fluoroscopic sign, which is naturally the obliteration of the normal auricular and ventricular curves. Finally, the action of the diaphragm—its excursion—is observed, the right dome being normally higher than the left and its excursion more limited. The presence or absence of limiting adhesions and their extent if present are noted, and lastly, the presence of small quantities of fluid, not sufficient to attract attention in the first survey, will be noted by the obliteration of the angle between the diaphragm and the outer chest wall. The chance of error in the detection of even small amounts of fluid is slight if the observer is at all careful. Adhesions may simulate it, but their level does not vary with the acts of respiration. We have seen the dome of the diaphragm obliterated by adhesions which drew it to a tense horizontal line,

but here also is the absence of excursion, and there is little or no change by posture as would be the case with fluid. When the patient may not assume the erect posture, even momentarily, the detection of small amounts of fluid is less easy, but by alternate examinations with the patient now on the left side, and now on the right, a fairly accurate diagnosis even as to amount of fluid is usually obtainable.

In roentgenology many signs are plainly definite. Pleural effusion, pulsating aneurysm, diaphragmatic adhesions, cardiac displacement, foreign bodies—of these one speaks as confidently as he does of meeting an acquaintance. There is no ground for debate. But, is a given case just a slowly resolving pneumonia, or is there an engrafted tuberculosis present? Is this, evidently old, tuberculous lesion entirely quiescent, or are there areas of present activity, etc.? In a multitude of these and other cases only the most careful team work can give results at all to the patient's advantage.

An effort is made to present here cases of especial interest to the general practitioner, cases which may originate in any family practice. Particular attention is paid here to cases which are distinctly typical and to those in which the clinical diagnosis was quite disproved. The name of the clinician is omitted in some of these latter cases because of unpleasant inferences that might be drawn. These clinicians were men of more than average ability, and the errors are, I believe, unavoidable and furnish the strongest argument for the intelligent use of x-ray study in thoracic disease. By intelligent use is not meant that either the clinician or the roentgenologist shall accept the opinion of the other, but that the two by persistent "team work" shall arrive at a well-understood working basis.

CASE I (from the service of Dr. Arthur Newlin, Pennsylvania Hospital).—A. S.; female, æt. sixty-four. Diagnosis: pleural effusion. Family history, negative. Personal history, negative up to six months ago, when left breast was removed for a malignant growth. Began six days ago with shortness of breath, choking sensation, weakness, sweating, loss of appetite, dry cough, no chest pains.

*Examination.*—Right side, flatness with increased resistance; diminished vocal resonance and fremitus and breath sounds. Breath sounds over upper portion exaggerated and note slightly tympanitic. Lateral and anterior aspect of lower two-thirds of right chest shows many crackling râles, a few sibilant râles, bronchovesicular breathing. Left chest, negative.

Fig. 152: *x-Ray examination.* Typical, rather excessive, right-sided pleural effusion. This effusion and the shadow of the breast effectually block out all other detail. Normal lung

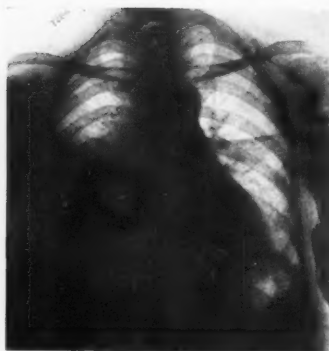


Fig. 152.

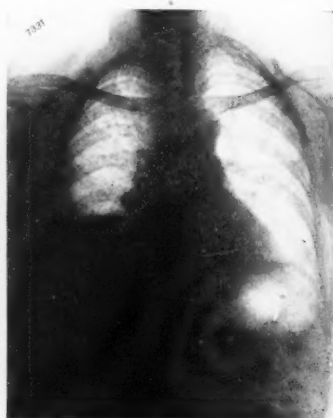


Fig. 153.

shadows above the effusion are accentuated, but not more than can be logically explained by the lung compression. There is a very distinctly thickened interlobar pleura on the left side. Any suggestion regarding the original lesion causing the effusion is quite impossible.

Fig. 153: Fourteen days later. The fluid does not reach nearly as high as in the first examination. Conditions are otherwise unchanged.

Fig. 154: Six months later. On the left side there remains unchanged the thickened interlobar pleura. There are also

dense pericardial diaphragmatic adhesions with marked abnormal density in the lower portion of both lungs and accentuation of the peribronchial shadows in the upper part of both lungs. These findings are not particularly significant of malignant disease. [In this case it seems quite probable that the effusion was not due to metastasis, as would naturally be suspected.] Figures 152 and 153 are quite typical of any pleural effusion which is not walled off and which is not accompanied by pneumothorax. This is an appearance very easily recognizable not only in plates, but in the fluoroscope, and in the fluoroscope the diagnosis can be almost instantly verified by changing the position of the patient. It is to be noted particularly that the contour of the



Fig. 154.

fluid does not assume a level as is the case whenever pneumothorax is present.

CASE II (service of Dr. Arthur Newlin).—D. B.; male; æt. thirty; Italian; laborer. Provisional diagnosis: acute pleurisy, chronic endocarditis. Family history, negative. Married three years; wife and two children living and well. There is occasional alcoholism, moderate use of tobacco; indoor occupation. No previous illness. Not predisposed to colds. Has had slight unproductive cough for a year or more.

Present illness began one day ago, with a series of chills; sharp pain in right side of thorax anteriorly, increased by deep breathing or coughing. Cough productive and toward evening

sputum became blood streaked. There was shortness of breath and fever.

Fig. 155: x-Ray examination. Plate taken with patient reclining. Large right-sided pleural effusion. Plate typical in the reclining position.

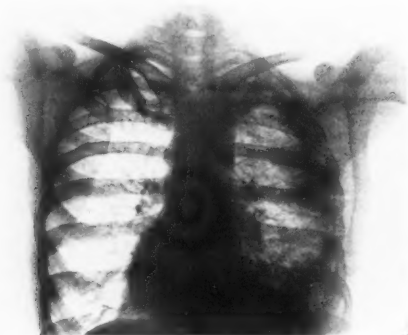


Fig. 155.

Fig. 156: Two days later, after partial aspiration, during which air had been admitted to the chest. The patient im-

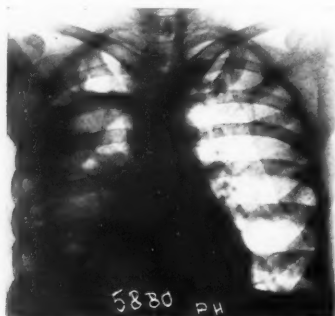


Fig. 156.

mediately previous to this examination had been reclining. He was placed in the erect posture and the plates rather hurriedly

made. Note three areas of pneumothorax each above a level area of fluid. This interesting condition was undoubtedly temporary and produced by placing the patient in the erect posture.

Fig. 157: Examination at the end of six weeks just previous to discharge shows some remaining accentuation of the hilus shadows, a distinctly thickened right interlobar pleura, but, on the whole, quite confirming the clinical appearance that the patient has practically recovered.

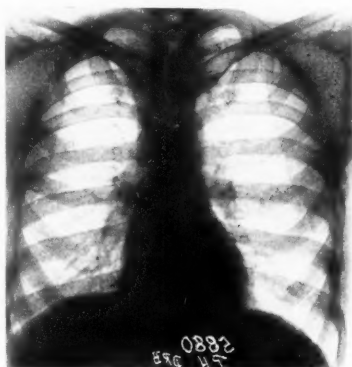


Fig. 157.

More or less closely associated with pleural effusion and empyema unrestricted on one side of the thorax are the cases of encapsulated or walled-off empyemas.

CASE III (from the service of Dr. Arthur Newlin, Pennsylvania Hospital).—A. B.; male; æt. forty-four; oyster opener. Provisional diagnosis: acute pleurisy. Complained on admission of sharp pain in right side of chest, increasing in severity on both inspiration and coughing. Family history, negative. Is a moderate drinker; uses tobacco excessively; has been an oyster opener for twenty years. Had typhoid fever at the age of nineteen; several infections of the hands and some chronic arthritis. Present illness began suddenly twenty-four hours ago with

severe pain in lower right side of chest, mostly anteriorly. Had slight cough for past two or three days only, non-productive; no blood spitting. There has been no loss of weight. During the following two weeks the subjective symptoms decreased, but there was very rapid loss of weight.

Fig. 158: Examination shows a typical, parietal encysted empyema, probably originating between the upper and middle right lobes and gradually assuming the pendulous position here shown. The patient was at once drained through a resection of the sixth rib. A large quantity of thick light green pus of foul odor, but in which the colon bacillus was not detected, was

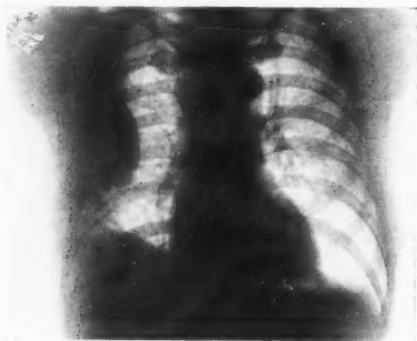


Fig. 158.

removed. Recovery was prompt and complete. Surgeon, Dr. Richard H. Harte.

CASE IV.—W. C.; male; æt. forty; laborer. Provisional diagnosis: pulmonary abscess. Family history, negative. Personal history, typhoid fever at twenty-eight years; takes cold easily; denies venereal infection; uses whisky and tobacco to excess. Four months ago while at work sat down to rest, when he had a sudden coughing spell, which produced a hemorrhage of about a teacupful. Later there were two similar hemorrhages. During this time his weight has decreased from 172 to 135 pounds. He expectorates a large amount of greenish sputum; has no

night-sweats; appetite is good. Patient is pale, poorly nourished; decided clubbing of fingers; slight cyanosis of finger-tips; chest is flat. Expansion is equal anteriorly. Posteriorly, greater play of left scapula than of the right. Percussion gives slightly higher pitched note at right apex than at left. Note throughout rather woody in character, with impairment at right base extending around to right lower axilla. Vocal and tactile fremitus are normal. A few fine râles heard in extreme right apex, right base, and left lower axilla. Percussion note impaired at extreme right base posteriorly.

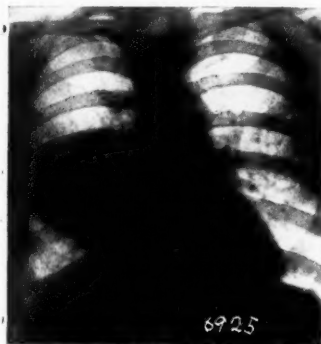


Fig. 159.

Fig. 159: *x*-Ray examination fifth day after admission. There is a large area of great density opposite the seventh interspace posteriorly, fourth interspace anteriorly, right side. This area is surrounded by an area of diminishing density which reaches to almost every part of the right lung. Considering the location and the rather sharply defined curved borders this would seem to be an encapsulated interlobar empyema rather than a lung abscess. There is some thickening of the parietal pleura at the level of and below this area.

Fig. 160: *x*-Ray examination fifteen days later. There is comparatively little change. The mass before reported seems a little smaller; the density of the surrounding lung, especially above the mass, a little less. The mass does not show any area

of lesser density, as would be the case if it were an abscess partly

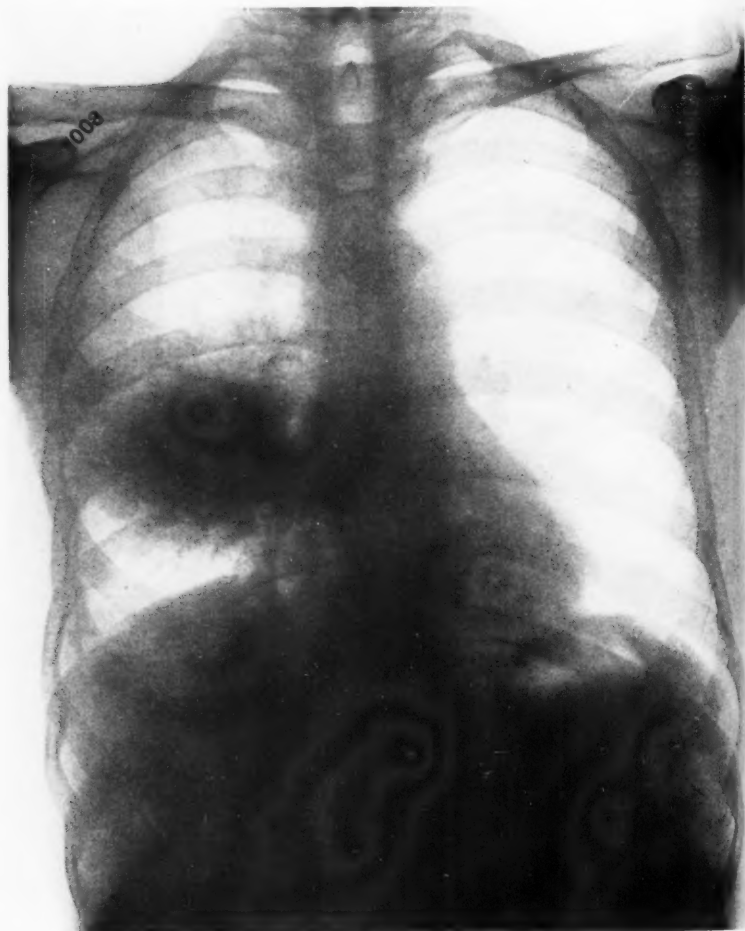


Fig. 160.

empty and filled with air. [During this period and the two weeks following the patient's condition improved perceptibly. There

was especially a great diminution in the cough and an almost complete cessation of the putrid sputum.]

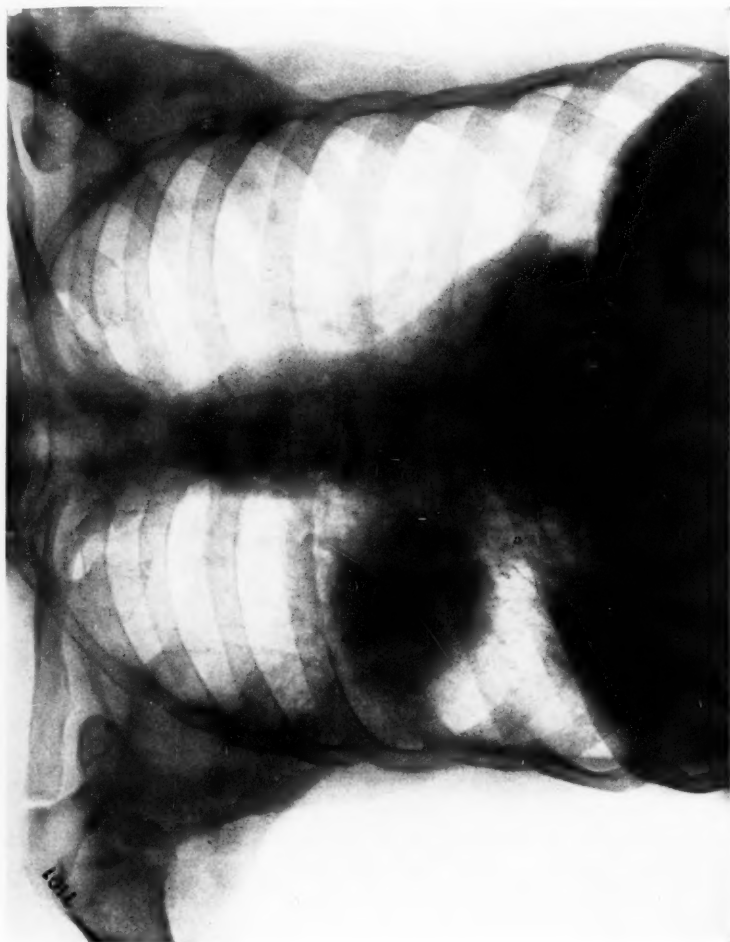


Fig. 161.

Fig. 161: A third examination three weeks later shows the mass somewhat smaller, the surrounding hyperplasia very defi-

nately reduced, and, by stereoscopic study, the mass is apparently equidistant from the anterior and posterior walls. The diagnosis of encapsulated interlobar empyema would seem to be positive. The patient was drained through an open incision three days after the last x-ray examination. It was necessary to go a considerable depth into the lung tissue, and the operative diagnosis between a lung abscess and interlobar empyema was not made. The patient developed pneumonia, apparently septic, on the tenth day after operation, and died on the thirteenth day subsequent to operation. Combining the clinical history with the x-ray findings it would seem that this patient had an interlobar empyema which was preceded by a lung abscess of some three or four months' standing.



Fig. 162.—(Reversed.)

CASE V (service of Dr. Arthur Newlin, Pennsylvania Hospital).—L. W.; female; colored; æt. thirty-seven; domestic. Family and personal history, negative. Has had for a few days pain in the upper right chest, some dyspnea, loss of appetite, general malaise. By examination the mediastinal dullness extends some three fingerbreadths to the right, and in the same area there is diminished breath sounds, vocal resonance, and vocal fremitus.

Fig. 162: x-Ray examination shows typical walled-off mediastinal empyema which was drained by open operation.

Operative diagnosis: apical empyema. Discharged cured. Surgeon, Dr. Richard H. Harte.

CASE VI (patient of Dr. George W. Norris).—Spontaneous pneumothorax. Male, aged about thirty-five years. Previous history of tuberculosis, with apparent recovery twelve years before.

Fig. 163: On the day previous to this examination patient awoke in the morning with a feeling of distress in the right chest and slight shortness of breath; otherwise without symptoms.

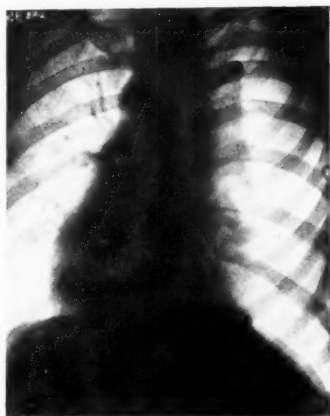


Fig. 163.

This plate was made after clinical diagnosis of pneumothorax. Note the partially collapsed right lung showing adhesions at the apex which prevent its entire collapse, showing also very plainly division between the middle and lower lobes.

Fig. 164: The second examination five days later shows considerable expansion of the collapsed lung, but a hydropneumothorax. Examination of similar cases with fluoroscope is highly satisfactory, the slightest movement of the patient being shown to cause waves in the fluid, making its recognition prompt and easy.

What is much more surprising is the rather more than occa-

sional error in which a collapsed lung with pneumothorax is mistaken for a large pleural effusion. This, in our experience, has occurred too frequently to be counted as an accident or explained solely by the negligence of the clinician. It apparently depends upon the same principle of sound modification as does the change in the tone of a drum when its air vent is closed. The ruptured lung, acting with a valve effect, permits overdistention of the involved side of the chest.



Fig. 164.

As in this case (Fig. 165, from the x-ray service of Dr. W. F. Manges, Jefferson Hospital), where the left lung is shown totally collapsed in a position in which it might almost be mistaken for an enlarged shadow of the left auricle, the heart and aorta are shown markedly displaced to the right. The collapse of the lung is confirmed by the total absence of lung detail throughout the right chest which is especially marked when compared with the increased lung detail in the right chest. In this patient there was, clinically, decreased expansion of the left side, diminished vocal fremitus amounting almost to absence, accompanied by tendency to hyperresonance with slightly increased fremitus over

the right side. The percussion note on the left side somewhat

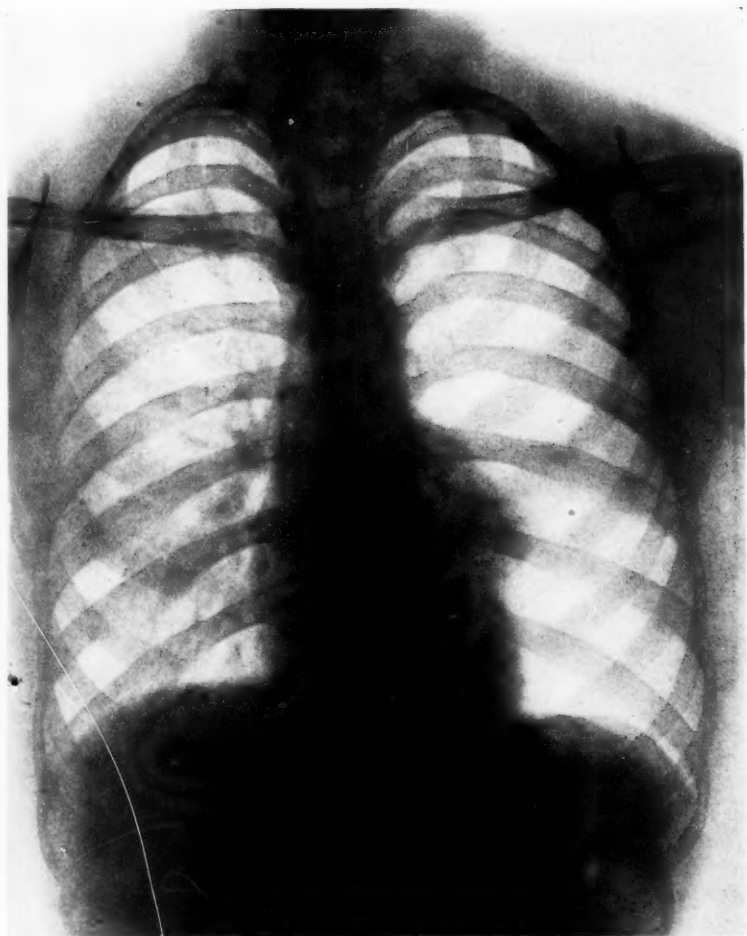


Fig. 165.

impaired; breath sounds almost entirely absent. Breath sounds on the right side harsh, with occasional râles.

It is not my purpose in this clinic to enter into the discussion, pro and con, of the x-ray diagnosis of pulmonary tuberculosis. A single case is shown as an example of the many in which the progress of the disease, while extensive, has been so insidious as to escape the notice of the patient or his family. Frequently, as in the present case, the progress of the disease as shown by x-ray is quite out of proportion to the clinical findings.

CASE VII (service of Dr. Arthur Newlin).—This patient, K. I., female, eleven years of age, was admitted on account of abdominal pain. The father died of tuberculosis. Mother, one brother, two sisters are well. Pertussis three



Fig. 166.

years ago. One short attack of bronchitis a year ago. Otherwise well until present illness. For the past three weeks ill with abdominal pain, especially after eating. Nausea, vomiting, headache, cough for the past week; slight expectoration. Examination of lungs anteriorly: Impaired note at both apices and below clavicles to second interspace; increased tactile fremitus, exaggerated vocal resonance; prolonged high-pitched expiration; no râles. Posteriorly, dullness on right over summit to 15 cm. downward. Tactile fremitus increased; prolonged high-pitched expiration; egophony voice sounds. Left side, posteriorly: impaired note at apex and 10 cm. downward.

Over summit, prolonged high-pitched expiration; occasional crackling râles.

Fig. 166: Extensive increase in density throughout both lungs. This is at a maximum on the left side between the fourth and sixth ribs in the axillary line, and there is rather less density shown in the lower and external portions of the right lung. Running from the hilus toward both apices are cavities which have more an appearance of bronchiectasis than necrosis. The examination is highly characteristic of an advanced tuberculosis of comparatively slow growth. [This finding was borne out by the further clinical history and the death of the patient.]

One of the most frequent metastases of sarcoma is metastatic sarcoma of the lung.

CASE VIII.—This case is exceedingly typical, not only as to the x-ray finding, but as to clinical symptoms. G. P., male; æt. twenty-two; Italian, laborer; admitted October, 1913, with sarcoma of the left forearm. Had been operated January, 1912, in Italy. The tumor began to grow again; the arm became painful, but on admission the growth was limited to the forearm; the elbow was not involved; there was no involvement of the axillary glands, and removal was done without amputation. Pathologic diagnosis: spindle-celled sarcoma. Treated with Coley's fluid for five months; discharged; returned for Coley's fluid and observation. Readmitted May, 1914, with severe pain in forearm. There was a return of the local growth, but no indication of metastasis. Lung examination was apparently negative, though there is a note made of slight mucopurulent expectoration. The patient was given Coley's fluid and x-ray treatment.

In August, 1914, the arm was amputated at the shoulder. Pathologic diagnosis: round-cell sarcoma. Patient was placed under postoperative x-ray treatment. During the next six months the patient did odd jobs around the hospital, running the elevator, doing orderly work, etc. He was in constant communication with the staff, and was more or less of a hospital pet. There was no return of the growth locally. The

patient was apparently well, and made no complaint until the day before this examination.

Fig. 167: At this time he complained of rather severe pain in the chest which led to the clinical examination followed by x-ray study. There are to be noted three very large masses in the left chest, one of which overlies another, and there are to be counted in the original plate thirteen separate foci of metastases. When the condition and its inevitable result were explained to the patient he returned to Italy. This case is particularly typical in the large degree of involvement without subjective signs.

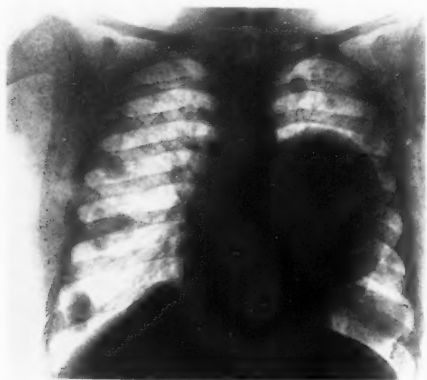


Fig. 167.

CASE IX (service of Dr. Arthur Newlin, Pennsylvania Hospital).—In some respects this case is similar to the preceding. F. P., æt. twenty-two years; Italian. The patient had had amputation of right leg above the knee eleven months previously for osteosarcoma. One month after this operation right inguinal glands were removed, but no metastases found. The present trouble began a week ago with chills, fever, swelling of hands, arms, legs, and with aching pains in these parts. Sharp pain in left chest, slight pain on the right side; no dyspnea, no palpitation; some cough at night; no expectoration, no hemoptysis. The patient states he has lost considerable weight; exact amount

not known. Has never noticed the change in fingers (clubbing) until pointed out to him. Lung examination: Expansion fair and equal. Vocal fremitus good, except in right axilla below sixth rib, and posteriorly in left below inferior angle of scapula. Percussion over right lung negative. Left lung anteriorly, resonant with dullness below seventh rib in left axilla; dullness below inferior angle of scapula posteriorly. Breath sounds and vocal resonance decreased in these areas.

Fig. 168: x-Ray examination shows pleural effusion on the left side reaching to the third rib anteriorly. Above this there

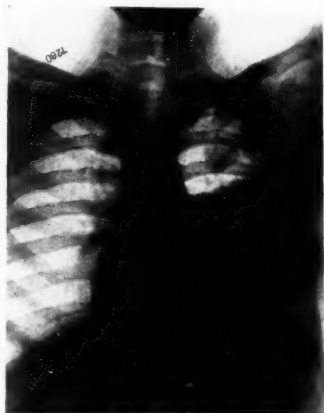


Fig. 168.

is a mass about  $1\frac{1}{2}$  inches in diameter underneath the anterior end of the second rib. This is quite typical of secondary lung sarcoma with effusion. Examination was made of all the long bones in the body with the result practically identical with that of the right hand.

Fig. 169: Periosteal proliferation is to be noted in every one of the long bones, except only the distal phalanges. The same condition was found in the feet, neither the carpus nor the tarsus being involved. There was no perceptible involvement of the cranial bones. During the remainder of the patient's

life, about six weeks, there was no apparent increase in this condition, nor was any bone change noted at all typical of sarcoma. Clinically, a case of osteo-arthropathy pulmonique (Marie). The very unusual feature was the apparently rapid development of this condition, previously reported cases being of slow development and long duration, apparently always accompanied by chronic pulmonary disease.



Fig. 169.

In conclusion I wish to present briefly two cases as typical of a considerable and rapidly increasing number of a condition usually diagnosed as chronic bronchitis, sometimes as incipient tuberculosis, but really due to unsuspected foreign body. For the privilege of exhibiting these cases and as well, indeed, for the privilege of examining all similar cases which I have seen, I am indebted to the courtesy of Dr. Chevalier Jackson.

CASE X.—V. M. S., female; æt. thirty-eight; eight years ago began to be troubled with cough; once a week would expectorate a mass of material the size of a pea, cheesy consistency, greenish-brown in color, and of foul odor. She was thoroughly examined in a western city; no evidence of tuberculosis was found; the sputum was examined. The patient led an outdoor life for the next two years. It was then necessary to have a perineorrhaphy

performed. The patient went to her home in an eastern city.

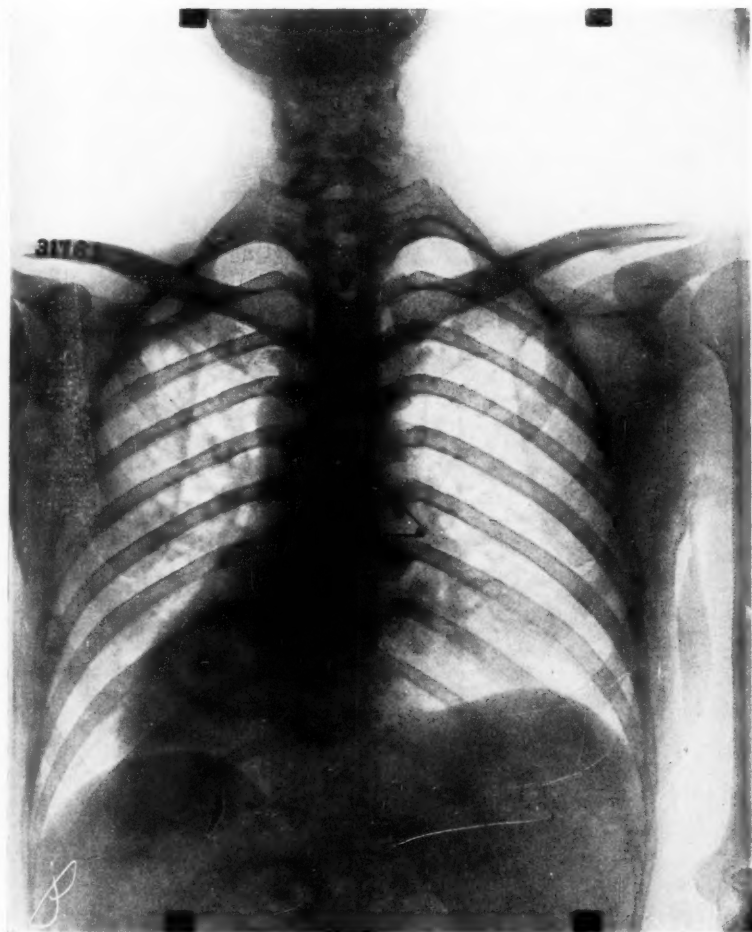


Fig. 170.

where, in the course of the general examination, an x-ray study of the chest was made, and a report given of an open safety-pin

in the right bronchial region. She was advised to "leave the pin alone as long as it leaves you alone." The cough gradually increased in severity and frequency, and masses, as above described, were expectorated more frequently. In the latter part of May, 1918, the patient had the first pulmonary hemorrhage, and a week later a second. Hemorrhages occurred very frequently from that until the time of admission in August. The amount varied from 6 to 16 ounces. Her family physician considered the case one of tuberculosis, disregarding the history of the safety-pin in the lung except as a possibly aggravating factor. The patient was brought from Florida to Philadelphia for examination by Dr. Jackson. She arrived in a stuporous condition, coughing up blood-streaked sputum.

Fig. 170: This plate was made some six hours before the patient's death. Neither she nor her family had any knowledge of the circumstances of the aspiration of the foreign body.

CASE XI.—D. M., male; æt. eight years. When two and a half years old while seated at the dinner table he suddenly spat up a mouthful of blood. An attack of "bronchopneumonia" lasting one week followed. A severe cough productive of a purulent expectoration followed, and at irregular periods the child spat blood in amounts varying from a mouthful to sputum which was merely blood streaked. For about two years, or until aged four and a half years, these symptoms continued in great severity, finally decreasing and the hemoptysis ceasing. The cough, however, continued, and there was purulent expectoration at intervals. There has never been a septic temperature; no night-sweats, but the child failed to grow strong. Repeated examinations were made for tuberculosis. Reports upon the sputum were always negative. At about the middle of the eighth year the father of the child, a physician, made an x-ray examination of the chest and discovered a foreign body in the lower right lobe. Two bronchoscopic examinations under ether anesthesia were made at an interval of about one week. In both there was failure to secure the foreign body and the patient was returned to his home. Some four months after the last bronchoscopic examination the patient was referred to Dr. Jackson.

Fig. 171: At this time the x-ray examination showed a metallic foreign body in the base of the right lung. In the fluoroscope the diaphragm shadow was seen to rise so as to completely cover the foreign body and fall so as to completely clear it. The foreign body is slightly less than  $\frac{3}{4}$  inch in length and about  $\frac{1}{4}$  inch in diameter at its upper large and blunt extremity. From this it tapers to a rounded point. A guess was made that it was the steel tip which fits in a tubular umbrella stem. There



Fig. 171.

was strikingly little surrounding pathology to be seen—just a slight increase in density immediately around the foreign body. The body was removed by Dr. Jackson by oral bronchoscopy under fluoroscopic control. The recovery of the patient, as in every one of these cases in which the foreign body has been removed, was prompt and complete.

These cases seem to be strikingly out of keeping with ordinary clinical experience. To the average layman or physician the aspiration of a foreign body in the lung

is an occurrence of such critical importance that the idea of an unsuspected foreign body in the lung seems almost preposterous. As a matter of fact, however, once the foreign body has passed through the trachea and into the bronchus, the relief from distressing symptoms is just as immediate and frequently as complete as if the foreign body had been expelled upward, and if x-ray examination of the lung had no other use than to reveal the presence of a foreign body in numerous cases of chronic bronchitis of unknown origin, its routine use in lung examinations would be fully justified.

**Conclusions.**—1. x-Ray study is exceedingly important in the general diagnosis of lung conditions.

2. The data yielded by this method are frequently such as can be obtained in no other way.

3. The valuable aid to be obtained by this method is, even yet, not generally understood, nor, so far as the average patient is concerned, generally used.

4. Its use in pleural effusion, whether free or walled off, is immediately and decisively satisfactory.

5. In tuberculosis and many other lung involvements, lesions, as revealed by x-ray, are very frequently more extensive than other clinical methods would indicate.

6. If for no other reason than to eliminate the question of unsuspected foreign body, this method should be used routinely in the clinical examination of the thorax.

7. The error of mistaking an excessive pneumothorax for a pleural effusion is not impossible even among clinicians of wide experience.

## CONTRIBUTION BY DR. S. D. W. LUDLUM<sup>1</sup>

PHILADELPHIA HOSPITAL

### PHYSIOLOGIC PSYCHIATRY

EVERY psychiatric clinic has had experience with cases typical of mental disorders which are generally considered incurable; when they do recover it is just as unexplainable as it is unexpected. We have been interested in cases of this sort, many having had all the signs and symptoms of dementia præcox; such cases as you would ordinarily decide would completely dement.

One of these cases, a young woman, began to improve phenomenally after beginning a treatment consisting largely of dilute hydrochloric acid, and within three months was totally well, and has remained so for two years. This girl had been having a continuously alkaline salivary secretion, occasionally an alkaline urine, and usually an acid stool. All these observations are quite opposed to the normal reactions, which are saliva alkaline before meals and acid afterward, urine acid, stool alkaline, blood nearly neutral. Following this case we have been making continuous observations upon newly admitted patients as to whether they were acid or alkaline in saliva, urine, blood, stool, and perspiration; and we have, I think, come upon some very interesting things.

In a large clinic like this, which is more of a clearing-house for the city's mental deficit than it is a treating hospital, it has appeared that if we disregarded the alcoholic and syphilitic, and tested those having the ordinary psychosis, we had frequently two groups—an alkaline and an acid group. This was not true of cases admitted where the condition had been of long standing,

<sup>1</sup> From the Psychopathic Department of the Philadelphia Hospital and the Laboratory of the Gladwyne Colony.

but was true of many acute cases. It seems that a disease having been existent for some time reaches a sort of pseudo-equilibrium; the observations made here have been based on the cases of short duration.

Quite early in this study the work of Loeb came to mind because of its close biologic relationship. In his book, "Dynamics of Living Matter," he shows the importance of a "regulator which is capable of keeping the solution in which marine animals live neutral." The cases that we have studied also have considerable analogy to the rapid modification of the behavior of fishes by contact with modified water; modified by altering its degree of acidity or alkalinity. Work of this sort has been done by Shelford and Allee in the Zoölogical Laboratory of the University of Chicago. Shelford says, "protoplasm and the plasmas of organisms possess a definite mechanism for maintaining approximate neutrality." Henderson says, "neutrality is quite as definite and quite as fundamental and quite as important a characteristic of the organism as is its temperature or osmotic pressure or, in fact, anything else we know of."

In the *Journal of Physiology* in 1890 we find that Roy and Sherrington give the results of their experiments upon circulation of the brain following intravenous injections of various substances. The substances having the most appreciable effect were acids and alkalies—acids raising blood-pressure and congesting the brain, alkalies lowering the blood-pressure and creating a brain anemia. The osmotic tension was undoubtedly different in each group.

There is quite enough biologic and physiologic data to assure one of the necessity of establishing neutrality or chemical balance in the nervous patient as a most fundamental procedure, and, following this, of establishing the physiologic balance. We have a number of such cases on record in these wards, and we become more and more impressed with the idea that mental disorder is but an expression of the visceral state. If this is so, it is quite surprising that so much has slipped past the internist. To illustrate, I shall give below in detail two examples—an acid and an alkaline case.

As a side light on the form that a psychosis may assume an article by Dr. Wm. B. MacNider appeared in "Science" last year concerning the influence of the age of an organism in maintaining its acid-base equilibrium. From the experiment cited it would appear that there is a definite association between the toxic effects of uranium and its ability to induce an acid intoxication; and the age of the animal very largely determines the rapidity of development and the severity of the intoxication. When animals of different ages were intoxicated by uranium the age of the animal was expressed by the degree of its ability to maintain the acid-base equilibrium. So it is possible that an acidosis or a pus-pocket in one individual gives an expression of a certain psychosis which in an older individual would produce a different psychosis.

The two cases which follow I consider typical examples of a departure from neutrality in opposite directions. One an alkaline state and the other an acid condition. Both cases made a prompt recovery when neutralizing substances were given as medicine, and the food selected to leave the appropriate acid end-products or alkaline residues as the case required.

CASE A.—A woman forty-seven years old, still menstruating, with the mental condition of excitement such as we see in manic-depressive insanity. She became sick rather suddenly, and went into this condition of manic symptoms. It was difficult to get her to sleep and more difficult to keep her in bed. The blood-pressure ranged about 190. She was in this condition for six weeks before we discovered that all her chemical symptoms grouped together. With litmus paper her saliva was invariably alkaline. The urine was very often alkaline on passing (she had no cystitis). The stool was often acid. The perspiration was sometimes acid, sometimes alkaline. The H-ion in the blood showed a figure indicating the low limit of normal, and the alkali reserve tests showed a high border of the normal. Van Slyke's  $\text{CO}_2$  test showed a high normal. The teeth were bluish in color, suggesting poor lime metabolism, and the lime recovered in the urine was in excess of normal; evidently she was not taking care of her lime.

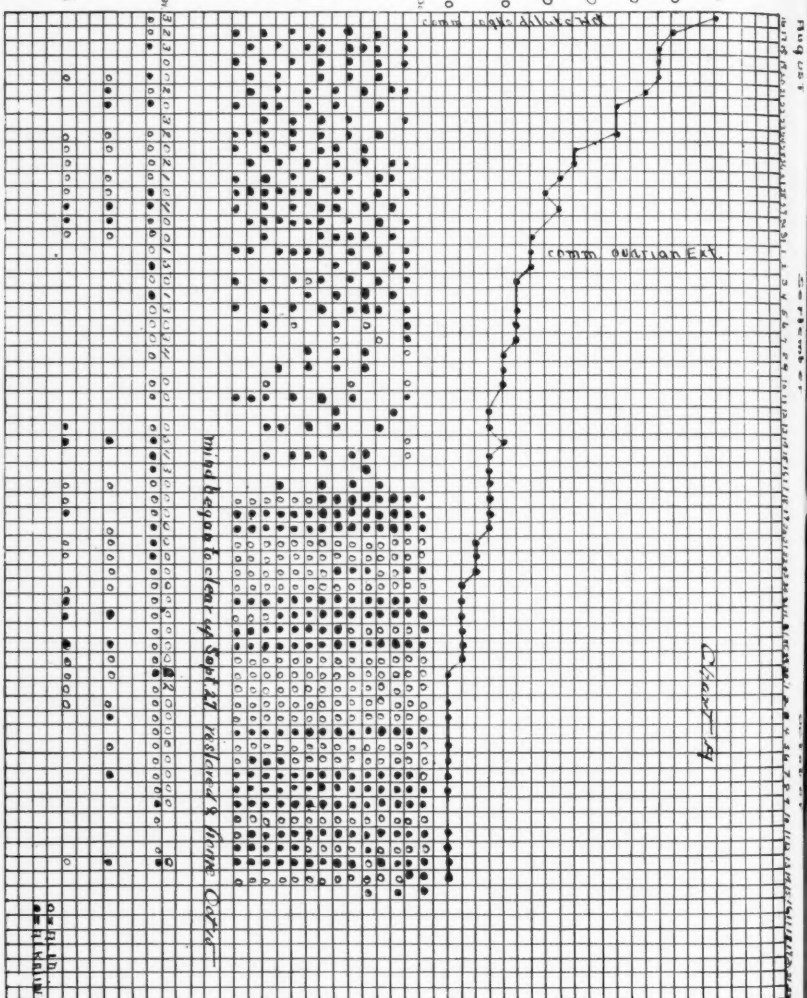
### Blood Pressure

# Saliva

### *Colicium in Urine* URINE

FECES

## PERSPIRATION



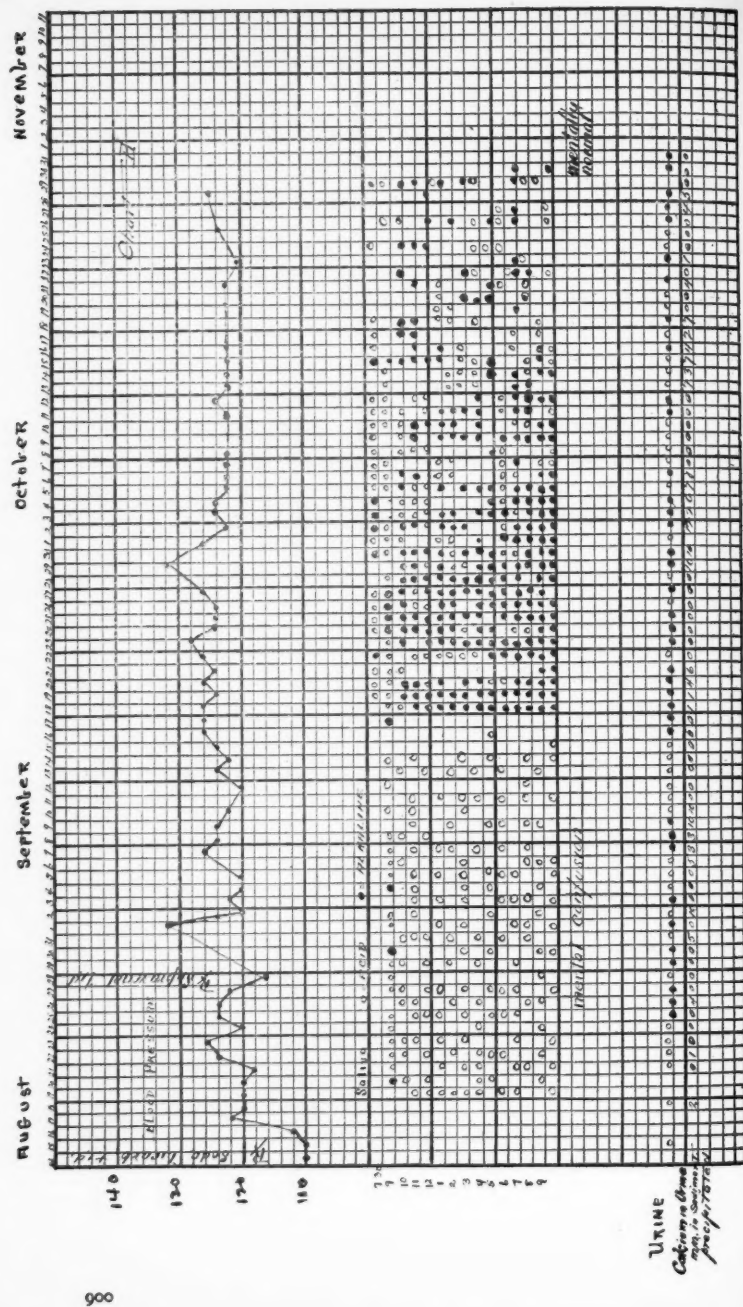
The chart indicates the case at the point where dilute muriatic acid was given as medicine, and it is interesting to note the progressive lowering of her blood-pressure. Tablets of ovarian extract were given because it has proved useful in the high blood-pressure cases with excitement. The chart shows in the course of three weeks the beginning of acid salivary reactions, until finally the observations demonstrated in the same day both acid and alkaline salivary reactions. At the same time it is noted that her blood-pressure had become normal, that the calcium was excreted in the urine to a less extent, and that the bowels showed less variations (normally the feces should be alkaline). The mental symptoms had all disappeared by this time and the patient was discharged.

CASE B.—A woman with the acid diathesis, with mental symptoms of confusion. She had been previously recommended for the asylum, but she has made a very good recovery. Her blood-pressure was low, the saliva invariably showed acid reactions with litmus. The urine was often alkaline, the stool usually acid. The H-ion showed a high normal content, the alkali reserve a low normal, the Van Slyke  $\text{CO}_2$  a low normal. The calcium excretion was limited, but came up as she improved. She was given sodium bicarbonate daily to establish neutrality, and for the physiologic symptoms was given suprarenal tablets, which raised her blood-pressure. When she showed in the same day acid and alkaline litmus reactions in the mouth, when the blood-pressure had come up, and her other body cavities were giving normal reactions, so that one might expect proper osmosis to take place, she became sufficiently well to go home.

Many critics say that litmus is not a delicate enough test, but it always tallies with the collection of dialized saliva tested against color tubes, and for clinical purposes it is quite sufficient.

The condition of caries in the teeth has little to do in determining the reactions, for in these cases the teeth were the same throughout the period of observation.

I think that the practising physician can do a great deal toward caring for his nervous patients and often prevent their insanity by seeing that the fluids, secretions, and excretions



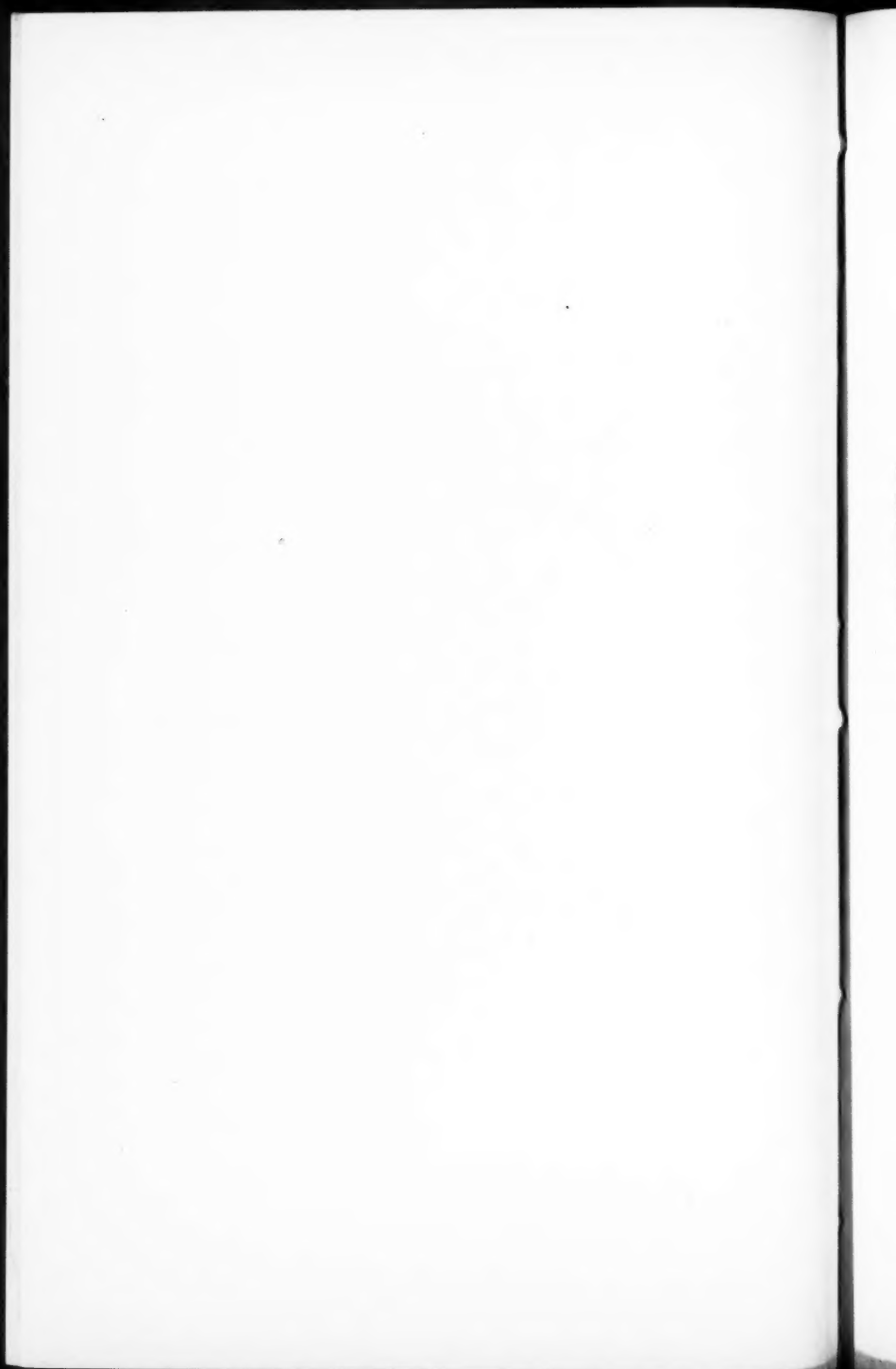
are showing their normal reactions. There is no question that osmosis can be greatly facilitated, and that patients are not so apt to become auto-intoxicated. The physiologic symptoms, such as blood-pressure, can be adjusted by using internal secretions in a therapeutic way, following institution of treatment tending toward the establishment of neutrality.

These two procedures we consider as fundamental in the treatment of acute insanity.

**Conclusions.**—I think the cases just described are quite typical of two groups that can be found in any acute ward. There are other groupings of physiologic, chemical, and psychologic symptoms. I am not saying that all insanity can be divided into acid and alkaline psychosis, but I do say that there is a stage in the early development of the disease when their metabolism expresses itself in this fashion. In the two cases described here the metabolism continued to so express itself.

The practising physician has from this point of view several avenues of approaching his mental cases before he decides to place his patients into specialized treating hospitals.

To summarize: The two types emphasized here are those with high blood-pressure, mental excitement, alkaline saliva, alternating acid and alkaline urine, perspiration, and feces; and those with low blood-pressure, mental confusion, and generally acid diathesis.



## CONTRIBUTION BY DR. DAVID RIESMAN

### INFLUENZA<sup>1</sup>—REMARKS UPON SYMPTOMS, PREVENTION, AND TREATMENT

THE influenza epidemic swept over Philadelphia as a plague of unheard-of virulence from about mid-September to the latter part of October. Only a fraction of the cases were reported, so that the total morbidity is unknown and figures are mere guesses. In the mortality statistics three terms appear in relation to the disease—"influenza," "pneumonia," and "bronchopneumonia." From clinical and pathologic evidence and from epidemiologic considerations we are justified in saying that all fatal cases of pneumonia, bronchopneumonia, influenza, and even of bronchitis occurring during the prevalence of the epidemic were one and the same disease. I believe, with Christian,<sup>2</sup> that all fatal cases of influenza were accompanied by pneumonia except those fulminating ones in which death occurred from pulmonary edema. From the clinical standpoint it was difficult to separate the severe cases of influenza from those of pneumonia. Are they really distinct? To me it appeared as if the pneumonia was not an accidental complication, but an integral part of the epidemic disease when assuming a severe form. The undoubted occurrence of non-pneumonic or apneumonic influenza naturally suggests the idea that in the pneumonic form we may have been dealing

<sup>1</sup> Between September 23d and October 29th 47,012 cases of influenza were reported, indicating that about 2.5 per cent. of the population was attacked. I am inclined to think that this is far below the true proportion. The number of deaths reported from pneumonia and influenza during the period between September 30th and October 31st was 12,032. The highest number of deaths from pneumonia and influenza alone in any one day was 711, on October 15th, which is more than 100 above the average weekly death-rate from all causes in Philadelphia. Nearly one-third of the deaths occurred between the ages of twenty and thirty.

<sup>2</sup> Jour. Amer. Med. Assoc., November 9, 1918, p. 1565.

with a process in which bacteria other than those causing the influenza were at work. It might be conjectured that the influenza germ, whether it be the Pfeiffer bacillus or something else, greatly favored the symbiotic growth of other pathogenic organisms possible—the pneumococcus, streptococcus, staphylococcus. Be that as it may—I do not know—but for me the clinical picture of influenzal pneumonia was distinct from that of other forms of pneumonia; was that of a definite entity, as much so as scarlet fever, in which an unknown virus and the streptococcus are probably responsible for the clinical picture.

The mere fact that pneumococci, streptococci, or staphylococci are found in the lung in cases of influenzal pneumonia does not prove that they are the cause of the pneumonia. We must remember that the lungs are in free communication with the air and with the germ-laden oral and nasal cavities. After every violent cough there is an involuntary aspiration which must carry many bacteria down into the deeper parts of the lungs. In typhoid fever and in other specific diseases of the intestine many bacteria are present in the bowel contents besides the specific micro-organism. They may play a rôle, but they are not the essential cause of the disease.

Much mystery surrounds the influenza bacillus. I think we may safely say that it was present in virtually 100 per cent. of the pneumonias of the epidemic. The failure of many observers to find it in the lung tissue or the sputum was probably due to imperfect technic. If consistent presence established etiologic relationship, then we should be in possession of the cause of the disease. But that fact constitutes only one of the four laws of Koch. Neither in man nor in animals has the disease been produced experimentally with a pure culture of the Pfeiffer bacillus. The serologic reactions obtained by Spooner, Scott, and Heath,<sup>1</sup> while interesting and important, do not permit far-reaching conclusions with regard to the causal relationship of the Pfeiffer bacillus. The question whether the Pfeiffer bacillus is the etiologic factor in influenza still remains. Opinion counts for nothing in answer to it, but definite facts we have not.

<sup>1</sup> Jour. Amer. Med. Assoc., January 18, 1919, p. 155.

Some<sup>1</sup> have searched for a filterable or ultramicroscopic virus, but in vain.

The beginning of the epidemic in Philadelphia gave no clue as to the terrible scourge it was to become. I remember that one morning in the middle of September Dr. Wilmer Krusen, Director of Health, called me up on the telephone and asked whether I had seen any cases of influenza. Having just returned from my vacation, I had seen none. The very next day I saw two, a physician and a woman in my neighborhood. Both developed pneumonia, the physician, of the bronchopneumonic type; the woman, of the lobar type. Both recovered. Very soon the increase was fast and furious, and the majority of the cases I saw—chiefly in consultation practice and among the nurses and doctors in the Philadelphia General Hospital—were of the pneumonic type.

In an overwhelming number of cases the pneumonia arose early in the disease, sometimes in the first twenty-four hours, sometimes not until the second or third day. Percussion seemed to give more help in the early stages than auscultation. Usually some impairment would be found over a small area in the post-axillary line and about the angle of the scapula. At first this was most frequent on the left side, but later the inequality between the sides disappeared. Primary apical cases were exceedingly rare, but the apex was quite often found involved secondarily. On auscultation over the suspected area one usually could hear a shower of fine crackling râles; sometimes only at the end of a vigorous inspiration. One could predict with fair assurance that within twenty-four hours the suspected area would be dull and the seat of bronchial breathing. Sometimes the consolidation would spread with great rapidity, usually involving the whole lower lobe of the lung posteriorly and rapidly creeping forward toward the sternum. Exquisite dry bronchial breathing and bronchophony could be heard over such an area. In many instances the percussion note was as flat and resistant as over a large effusion. Nevertheless, exploratory puncture

<sup>1</sup> Nuzum, Pilot, Stangl, and Bonar, Jour. Amer. Med. Assoc., November 9, 1918, p. 1562.

rarely revealed more than a small amount of clear or slightly blood-tinged fluid. In the majority of severe cases a little patch of consolidation could be found in the opposite lung, generally near the vertebral border of the lower scapula or in the post-axillary region. At times the pneumonia was almost as extensive in the secondarily involved lung as in the other. Some degree of pulmonary edema, shown by diffuse moist râles, was often evident and almost never lacking in the bad cases. In cases proceeding to resolution, the pulmonary signs passed through the stages with which we are familiar in the course of ordinary lobar pneumonia.

Empyema was surprisingly rare compared with the earlier pneumonias of the army camps in this country. I was also impressed by the infrequency of pleuritic symptoms in general, so common in pneumococcic pneumonia. In a few cases, however, there was intense pain along the margin of the ribs and over the lower chest which could be interpreted only as due to diaphragmatic pleurisy.

Before leaving this phase of the subject I want to reiterate what I said with regard to the value of *percussion* in the early detection of pneumonia. I take it that the slight dulness before much else is demonstrable is due to congestion, to overfilling with blood of the part of the lung that is going to be inflamed.

The bronchopneumonic cases were in some respects a revelation to many of my colleagues and to myself. It was unusual in our former experience to find bronchopneumonia at the age period the epidemic selected—young, healthy adults from twenty to thirty-five. Hitherto pneumonia in these had been usually of the lobar type, barring the measles pneumonia among the soldiers in the army camps. Now we were confronted with a distinctly new picture. The patient had severe cough, thin, fluid, salmon-colored expectoration, high fever, dyspnea, cyanosis, râles over the chest, although often more marked over one lower lobe, with shadowy impairment on percussion, or distinct patches of dulness usually just inside the lower scapula. There was little difference in the general behavior of the two types of cases, although I carried away the not very sharp impression that

the bronchopneumonic cases did better than those of the lobar type.

Epidemic influenza usually *begins* rather suddenly, with general malaise, pains in the legs, head and eyeballs, and in the back, the last at times as severe as in smallpox. Distinct chills are not very common. Chilliness, however, was frequently complained of. Sneezing and other catarrhal symptoms were conspicuously rare. *Cough* was at times present from the first moment; sometimes its appearance was delayed for two or three days. It was variable in intensity; in some cases, however, it attained a violence scarcely matched by whooping-cough. The *sputum* varied very much, sometimes it was a glairy mucus, as in ordinary bronchitis, frequently it was a thin, reddish or salmon colored fluid; at times distinctly rusty and even tenacious, as in ordinary pneumococcic pneumonia. In a few instances it was of the prune-juice character. No conclusion of special value could be drawn from the color of the sputum, but in practically all cases in which blood was present one could demonstrate the existence of bronchopneumonia or lobar pneumonia. The *temperature* was usually high ( $103^{\circ}$  to  $104^{\circ}$  F.) and hyperpyrexia was common. While true crisis occurred in some cases, the majority of those ending favorably terminated by rapid lysis. An interesting feature was the comparative slowness of the *pulse* in the first few days of the disease. It was not uncommon to find a temperature of  $104^{\circ}$  to  $104.5^{\circ}$  F., with a pulse of 80 to 90. In many cases with extensive lung involvement the respirations were also disproportionately slow. In the later stages the pulse usually became rapid and dicrotic and the respirations mounted to 50 or 60.

The *blood-pressure* showed one peculiar feature in a fair proportion of cases: a very low diastolic pressure, such as is seen in aortic insufficiency. In the majority of cases Gibson's rule was verified. Aside from toxic myocarditis, cardiac complications were rare; endocarditis was exceedingly uncommon; pericarditis occurred in several instances.

Of *gastro-intestinal symptoms* the most striking were nausea and vomiting, which in some cases attained such a degree that

everything else was overshadowed. The vomitus was often of a coffee-ground character. Bile was rarely present. The gastric symptoms were evidently toxemia, probably caused by an effort at elimination through the gastric mucous membrane, and usually yielded to treatment within twenty-four to forty-eight hours. Abdominal *tympany*, as in ordinary pneumonia, proved a distressing symptom. When marked, the outlook was usually bad. The bowels themselves gave little trouble and were either moved spontaneously or easily by enemas or mild laxatives. Diarrhea was occasionally noted; it was either (like the vomiting) a sign of toxic irritation or could be attributed to the free use of enteroclysis. Jaundice was exceedingly rare, quite unlike our experience in old-fashioned pneumonia. In a few cases the liver was distinctly enlarged, but there were no signs of cholecystitis. Pain and tenderness in the right iliac fossa suggesting appendicitis were noted in several instances, but in my personal experience none of these cases was appendicitis; virtually all were examples of pain referred from the chest. I have heard, however, of true appendicitis complicating influenza.

In only one case was I able to feel the spleen easily. At times I inferred from percussion that it was enlarged, but it is well known how unreliable percussion is in determining the size of the spleen.

*Nervous Symptoms.*—Nothing illustrates more strikingly the profound toxemia of the disease than the nervous symptoms in severe cases. Insomnia was very common and intractable. Muttering or active delirium was also frequently present. One of my patients, while he had a temperature of between 105° and 106° F., overpowered two nurses and jumped out of bed. It was common to see a typical typhoid state: cracked lips, dry, glazed, brown, shrunken, tremulous tongue, carphology, muttering delirium, incontinence of feces and urine, coma vigil. Subjective complaints were often strikingly in abeyance, and some of the worst cases with extensive lung involvement, deep cyanosis, and persistent cough never complained at all. Such patients would often answer the query "How do you feel?" with "Fine! splendid!" In time I began to dread such answers as of bad

omen. In a few cases the picture of meningism was noted, but lumbar puncture showed nothing more than an increased amount of water-clear fluid. In this type, however, the mortality was very high. Headache, while severe in a few cases, rarely lasted long, and frequently was entirely absent. In one case, that of an intern in the Philadelphia General Hospital, a peculiar state of disorientation remained for some time after convalescence was established, but cleared up eventually. Dread and apprehension seemed to me in a few instances to have a share in the fatal termination. Thus several nurses, who had seen companions die, immediately on falling ill gave up all hope of recovery, and it was well nigh impossible to imbue them with a fighting spirit. It is true not all of these died.

*Urinary Symptoms.*—A striking feature of the majority of cases, both good and bad, was free diuresis, probably connected with the abundant water intake and fairly efficient kidney function. Albuminuria was commonly present; no more, however, than is seen in other acute infections; but in a few cases a true hemorrhagic nephritis, with marked albuminuria, dark and light granular and cellular casts, and abundance of free blood was noted. Even in some of these the quantity of urine kept up very well. A not infrequent symptom, sometimes overlooked, was retention of urine with great distention of the bladder.

*The Skin.*—Herpes was rare, though when steadily looked for a single vesicle or crust, or perhaps two, could be discovered in a considerable number of cases. I did not see any other rashes except in one or two instances a rather bright general erythema. An occasional bed-sore was observed.

*Subcutaneous Emphysema.*—One night I was called in consultation to see a young girl of twenty who had been ill for three or four days. I found an extraordinary picture. Her neck was so swollen as to be practically obliterated, the swelling extending into the supraclavicular spaces and out toward the shoulders and upward toward the face. The girl's voice was scarcely audible. She was moderately cyanosed, had a severe cough, and an extensive bronchopneumonia. On palpating the neck structures a most exquisite crackling was detected. It is

not easy to explain the origin of the subcutaneous emphysema in this case—several have been reported in the accumulated literature—but it would seem probable that rupture of the lung occurred during cough at a point of adhesion to the mediastinal structures, as there was concomitant pneumothorax.

*Cyanosis*.—No symptom was so striking both to the physician and the layman and so distressing in its ominous significance as cyanosis. In mild degrees it was confined to the lips, the tips of the ears, and the finger-nails. In severe cases it was general over the face, the trunk, and the extremities. The lips and mucous membrane of the mouth often had a maroon color, while the face and neck were of a livid bluish-black hue. Coughing and other muscular effort deepened the cyanosis. As in the cyanotic cases the heart was often beating vigorously and the pulse-pressure was well maintained, the cyanosis could not be attributed to circulatory failure, nor did it seem to bear an unmistakable ratio to the amount of lung involvement. It was distinctly proportional, however, to the toxemia. For that reason and also because of its color it suggested an altered state of the blood analogous to that of acetanilid-poisoning. This led me to use the term *hemocyanosis* in speaking of it. No definite studies on the nature of this hemocyanosis exist, although Synnott and Clarke,<sup>1</sup> on the basis of some spectroscopic tests, concluded that there was no methemoglobinemia.

*Nosebleed* was very common and often profuse to the point of alarm. In previous epidemics of influenza this symptom was not prominent. Uterine bleeding, usually a premature menstrual period, was very frequent, but did not assume any serious proportions. Hemorrhages also occurred from the stomach in cases with severe gastritis. Blood in the urine was often detected microscopically, but visible blood was rare, occurring in only one or two of my cases. It is scarcely necessary to refer again to blood in the sputum. It was very common. At times there was a distinct hemorrhage, as if from a pulmonary infarct.

The *blood counts* in my cases corroborated the findings of others as to the existence of leukopenia in the early stages. In

<sup>1</sup> Jour. Amer. Med. Assoc., November 30, 1918, p. 1816.

the later stages the counts often rose to 12,000 or 13,000, sometimes higher, in the presence of a complication such as otitis media.

Phlebitis, a not infrequent complication of ordinary lobar pneumonia, was rare in the contagious pneumonia of the epidemic. I can recall but one case.

*Pregnancy.*—When the disease occurred in a pregnant woman in Philadelphia it nearly always spelled death. Of those attacked at the height of the epidemic only a vanishing number recovered. It seemed to require but a short exposure for the pregnant woman to get the disease. I remember a particularly tragic case I saw with Dr. I. V. Levi. A young man came down with a severe influenzal pneumonia; his wife, who was in the seventh month of her pregnancy, was immediately urged to leave the house. This she declined to do. We therefore strictly forbade her going into the sick room. She did so, however; promptly came down with pneumonia, and died in four or five days, while her husband was slowly recovering after a desperate attack of bilateral pneumonia with extremely severe gastric symptoms and perfectly horrible coughing fits. One pregnant woman, whose husband had a virulent influenzal pneumonia to which he succumbed, was isolated as soon as practicable and escaped, one of the few exposed ones to do so.

Most of the pregnant women aborted. At first I hoped that this might be an advantage, but it did not prove so. The majority died with symptoms of profound toxemia, cyanosis, typhoid state, and extreme tympany. In a number of cases the question arose as to the advisability of inducing labor. My advice was against it, and this was likewise the opinion of obstetricians called in consultation in some of the cases seen by me. As I have already stated, a large number of women had a premature menstrual period at the height of the disease. It did not seem to me that this had any influence in one way or another on the course of the infection.

*Atypical Cases.*—1. *Fulminant pulmonary edema* with death in twenty-four hours or less. These cases were fortunately rare, and seemed to occur in such persons as were exposed to con-

centrated infection and at the same time exhausted by overwork. These patients drowned in their own juices before pneumonia had time to develop. The poison behaved very much like some of the poison gases used in the war.

2. *Prostration Cases*.—In several adult patients I observed a condition that may be described as follows: A slight subfebrile state, with temperature a degree or more above the patient's normal—which in many was 97.4° to 97.8° F.—malaise becoming more marked in the late afternoon, and a physical prostration, and at times also a mental lassitude, quite out of proportion to the demonstrable signs. In some cases a cough existed and gave a ready clue to the infection. I believe these cases illustrate the occurrence of influenza in persons having a moderate degree of immunity.

3. *Hemorrhagic Nephritis*.—As a part of a severe influenzal infection this has already been described. It occurred, however, as a seemingly primary febrile disease, especially in children. While difficult of proof, the view that, in the absence of scarlet fever or other clearly demonstrable cause, it was an influenzal effect seemed justified.

One feature of epidemiologic interest should be mentioned, namely, the rarity of other acute infections during the reign of the influenza. It is possible that this rarity was only apparent and produced by the fact that my services were almost completely monopolized by influenza cases. Nevertheless, I am practically convinced of the absence of tonsillitis, ordinary acute colds, and erysipelas during the epidemic.

**Treatment.**—Our experience in Philadelphia taught us a painful lesson. We were unprepared for the great emergency as regards doctors, nurses, hospital space, drugs and supplies, coffins, etc. One of the most distressing features was that many patients suffered and died without medical aid. The doctors worked as they had never worked before, but they were unable to answer more than a fraction of the calls. The lessons learned I fear will be forgotten, but they ought not to be. I want to point out some of them, for to be guilty of unpreparedness in the future would be a municipal crime.

1. *The Zone System for Doctors.*—During the epidemic it often happened that at the time when many sick were unable to get a doctor, two or three or four might be visiting patients in the same block. I remember one day at the height of the epidemic in one short suburban square there were four doctors' motor cars. One physician could easily have seen all of the cases in the square, while the other three, I calculated, could have seen forty-five patients in the city during the time consumed in making the trip. My plan is to zone the city and to assign to practitioners a definite district to be under their charge during a virulent epidemic. Judgment must, of course, be used; to the younger man a larger number of houses can be assigned than to the older one. Objection might be made that patients want their own doctors, but I found during the epidemic the people were glad to get anyone who could come soon enough and often enough. Nor does the plan prevent the physician from seeing his own patients, provided he has discharged his assigned duty. It may be that doctors will object to such a plan as I propose. If they do, they cannot be coerced, and the execution of it would have to be left to volunteers. The city, of course, could hire men, but if that is to be done then plans as to their number and distribution should be made so as to be ready when required by an emergency.

2. There should at all times be a census or *roster of nurses* in the hands of the Health Department. No single patient should have two nurses unless the need is certified to by the attending physician. The department should likewise have a record of all semitrained and volunteer nurses, and both they and the regular nurses should be provided with printed postal cards on which to notify the Health Department whenever they leave a case.

3. *The Mobilization of Social Workers.*—A complete registry of social workers should be kept in the offices of the Health Department, and the best possible use made of these valuable adjuncts to medical and charitable relief. In one of the sections of the city especially hard hit by the epidemic the people raised a fund and among other things hired two capable social

workers who ferreted out the very bad and neglected cases. For them they procured nursing and medical attention, and even arranged consultations between men of the neighborhood and consulting physicians.

4. The *druggists* were fearfully overworked and further handicapped by a shortage of much-used drugs. It was suggested by someone that physicians should agree upon stock prescriptions to be put up in bulk, and called for by patients before the visit of the doctor. The plan is of doubtful utility.

5. *Reporting of Cases*.—This is eminently desirable. It was neglected by doctors overwhelmed with work. Moreover, they saw no special rationale in the time-consuming procedure, as nothing was done.

6. *Emergency Hospitals*.—Plans must be made for the creation of emergency hospitals on short notice, and must, like all other plans, be ready in a pigeonhole, like the campaign plans of an army general staff. Only in that way can one administration benefit from the experience of another.

7. *Quarantine* is of doubtful utility in an epidemic of such terrible proportions. If it were to be done it would probably lessen the reporting of cases.

8. The *closing of public places* seems to me a wise measure if judiciously enforced, but the matter is a difficult one, as is well brought out in the Report of the Special Committee of the American Public Health Association on Influenza.<sup>1</sup> In Pennsylvania the State Health Department closed schools, churches, moving-picture houses, theaters, and saloons. I hold no brief for saloons, but cannot help questioning the consistency of the state in closing them when factories, department stores, lunch counters, barber shops, etc., were kept open.

9. *The Wearing of Masks*.—This has proved itself a measure of unquestioned utility. It is, of course, necessary that the mask be properly made, rightly worn, and disinfected after use.

10. The *washing of the hands* is probably one of the most important measures in the prevention of the spread of the disease.

11. *Gargles and Mouth-washes*.—The use of mouth-washes and

<sup>1</sup> Jour. Amer. Med. Assoc., December 21, 1918, p. 2068.

gargles does not appear to me a sensible method of prophylaxis. I am aware that quinin in dilute solution, as shown by Kolmer, is pneumococcidal, but mouth-washes and gargles can never sterilize the whole oral and pharyngeal cavity and may deprive the mouth of its protective covering of mucus and saliva.

12. *Vaccination.*—As a direct protection against influenza the value of vaccination with a mixture of organisms seems in the opinion of most critical writers to be not yet established. My own experience is rather encouraging. Moreover, I have seen no harm come from it. The procedure may, in addition, have a psychologic value, in that it banishes in the timid the panicky dread of the disease that cannot but help to lower the resistance.

The work of Austin and Cecil and others seems to attest the value of vaccination against pneumonia. Whether it holds good against the influenzal pneumonia as well as against that due to Types 1, 2, and 3 of the pneumococcus is as yet unproved.

**Direct Treatment of the Disease.**—1. *Simple Influenza.*—The patient must go to bed immediately, must be isolated, and the attendants and family masked. The diet may be liberal—milk, milk-toast, cocoa, chicken or lamb broth, cereals, stewed fruit, junket, wine jelly, soft-boiled or poached eggs, orange-juice, pineapple-juice. Water should be given in abundance, a glassful every three hours; it may be given as lemonade, with grape-juice or with orange-juice. The bed-pan should be used whenever possible. Exposure increases the chances of pneumonia. The bowels should be moved in the beginning with citrate of magnesia. In the medicinal treatment I have used various combinations, without feeling that any is in the least a specific. A fairly satisfactory one is the following:

*R.* Quinin. salicylatis..... 2  
 Acetphenetidin..... 2  
 Pulvis ipecac. et opii (Dover's powder)..... 0.15.—M.

Divide into 12 capsules.

Sig.—One every three hours.

Or,

*R.* Acid. acetyl-salicylic..... 2;  
 Acetphenetidin..... 1.5;  
 Pulvis digitalis..... 0.1—M.

Divide into 12 capsules.

Sig.—One every three hours.

Or,

R. Acid. acetyl-salicylic..... 2;  
 Acetphenetidin..... 1.5;  
 Urotropin..... 2.—M.

Divide into 12 capsules.

Sig.—One every three hours.

The digitalis combination I have used in cases in which the pulse seemed to be soft from the very beginning. Dr. Edwin Zugsmith, of Pittsburgh, tells me that he has had very good results with a combination of sodium salicylate and urotropin.

2. *Severe cases*, by which I mean those with pulmonary involvement. The general principles are the same as those already given, but for this group of cases a nurse is almost indispensable. The doctor should invariably write out all directions in full, especially as in the stress of an epidemic he is not able to see his patients very often.

The so-called alkaline treatment was used by most physicians with whom I came in contact in the treatment of severe cases. I was in the habit of prescribing the following mixture:

R. Spiritus ammonii aromatic..... 5-10  
 Liquor. ammonii acetatis..... 30  
 Liquor. potassii citratis..... q. s. ad 150.—M.

Sig.—A tablespoonful in a glass of water every three hours.

In severely toxic cases the alkalies by mouth were supplemented by enteroclysis, with solution of sodium bicarbonate, a tablespoonful to a pint of water. In all severe cases digitalis was prescribed from the beginning, either the tincture of digitalis or digalen, occasionally digipuratum. In the later stages the dose of the tincture or of digalen would be 1 c.c. (15 minims); in the case of digalen to be given hypodermically every four hours.

In some cases we used strophanthin intravenously, 0.5 mg., and in every instance there was an immediate and perceptible influence upon the pulse which unfortunately was not sustained, and only postponed but did not avert the fatal ending.

In circulatory failure, shown by rapid, feeble pulse and pulmonary edema, atropin sulphate,  $\frac{1}{8}$  gr.; caffein sodium

benzoate, 0.12; camphorated oil in ampules, and, at times, adrenalin were used in addition to digitalis.

Throughout the epidemic glucose was extensively employed upon rather ill-defined indications, perhaps as much for general toxemia as for anything else. It was given by enteroclysis in 5 per cent. solution, and also intravenously. I cannot say that it has an outstanding value, but by supplying an easily metabolized material as well as a considerable amount of fluid it has possibilities of doing good.

With regard to alcohol, the last word has not yet been said. In the beginning of the epidemic little use was made of it. Later, somehow, we all fell into giving it quite freely, but I cannot say that I saw it do much good. Oxygen, though administered in enormous quantities, even to the point of wastefulness, seemed to have little influence upon the cyanosis, but did seem to lessen the dyspnea in some cases. Arsphenamine has been used empirically abroad. I have had no personal experience with it.

**Relief of Special Symptoms.—Vomiting.**—This frequently proved a tax on therapeutic resources. We found that it was best to give no food at all for six or eight hours and to apply a mustard plaster to the epigastrium, and then to begin with teaspoonful doses of buttermilk every fifteen minutes, increasing the amount and lengthening the interval as the stomach proved more retentive.

**Diarrhea.**—This very rarely required active treatment. When it did, two or three doses of bismuth subnitrate (0.5 to 0.6) proved effective.

**Abdominal Tympany.**—This symptom is met with under a variety of conditions, and is so distressing that it is well to have a routine treatment for it. If the patient has been taking milk, it has been my practice to cut off the milk and substitute broth or albumin-water for twenty-four or thirty-six hours. An asafetida suppository (0.6) and turpentine stupes to the abdomen prove helpful in the milder cases. If they fail, an asafetida enema (60 c.c. of milk of asafetida in a small amount of starch-water or plain water) may be used. If these measures fail,

pituitrin should be given hypodermically in doses of 0.5 to 1 c.c. It has taken the place of eserine, which was formerly used to stimulate intestinal peristalsis.

*Cough.*—Mustard plasters or dry cupping sometimes gave a measure of relief, but in the majority of bad cases an opiate was necessary.

*Insomnia.*—When the bromids—sodium bromid in doses of 0.6 to 1—failed to answer, my first resort was to chloralamid in 1-gram doses or to veronal in doses of 0.3. Failing in these, and they often failed, morphin had to be used.

*Pyrexia.*—Nothing special was done to combat the fever unless it was high. If it was persistently over 104° F., we resorted to sponging.

*Cyanosis.*—I have indicated in the foregoing that the cyanosis is chiefly a toxic symptom and that oxygen had little influence upon it. The general measures, such as the use of alkalies, perhaps of glucose, and the free administration of water, undoubtedly combated the toxemia and incidentally the cyanosis, at least in a measure. Several times I resorted to bleeding and the substitution for the blood of alkaline solutions, intravenously. But as all of these measures sometimes failed, I thought that transfusion, with or without preliminary bleeding, was worthy of a trial; but in the few cases in which this procedure was suggested it could not be carried out; in some, because the patient became moribund and the case hopeless before all the arrangements could be made; in one, seen with Dr. Reif, because the blood of every donor tried was immediately coagulated by the serum of the patient.

*Pleural Effusion.*—In the foregoing I have already stated that large effusions were rare, but even in a few cases with limited collections, the withdrawal of some of the fluid seemed to exert a most favorable influence upon the subsequent course of the disease. Thus, in the case of a doctor's wife, who had double pneumonia and seemingly had had a crisis, the temperature after a few days shot up again and persisted with rising pulse-rate and rapidly increasing prostration. Although the physical signs were those typical of consolidation—bronchial breathing

and bronchophony—an exploratory puncture revealed the presence of turbid fluid. A few syringefuls of this were immediately withdrawn, and thereupon, without any further interference, the patient began to improve and was soon on the high road to recovery.

**Convalescence.**—The physician must have definite rules for dealing with convalescent patients. Even after a mild influenza the patient should not be allowed to leave the bed until the temperature has been normal, or preferably slightly subnormal, for two full days. After that he should abstain from work until at least a week has elapsed. Severe cases require a prolonged convalescence. The patient must be kept in bed for ten days after the temperature has reached normal and abstain from undue activity for two weeks longer. It is best to send such patients away at the end of the ten-day period. For Philadelphians, Atlantic City offers all the desiderata for speedy recuperation.

During convalescence the heart sounds should be watched as to their force and rate. Absence of substance or strength and rapidity indicate a not completely vanished toxic myocarditis, which, in older persons at least, requires time for complete restitution to normal. Such persons must be kept in bed or in their rooms until the abnormal signs have entirely disappeared. Abundant food and tonic medication, for which I prefer elixir of iron, quinin, and strychnin, are indicated. If the appetite lags, it may be stimulated by an alkaline stomachic, as, for example, the following:

R. Sodii bicarbonatis.....	6
Tinct. nucis vom.....	6
Tinct. cinchonæ comp.....	60
Aq. cinnamomi.....	q. s. ad 90.—M.

Sig.—Teaspoonful in water three times a day.

I believe that the medical profession, mindful of the experience in the epidemic of 1890, is taking better care of convalescing patients than was the case then, and that we shall for that reason hear less of delayed fatalities.

As I write this article there is evidence that the epidemic is coming back on its second swing through the country. The

earlier cases are again mild, but during the last few days the dreaded pneumonic type, with profound toxemia, is again manifesting itself. The age incidence in this second visitation is somewhat different from that in the first. Children are more frequently attacked; also people of middle age and beyond. The children show a protracted fever of 103° to 104° F., with slight cyanosis of lips, delirium, cough, and a variable pulmonary involvement; a few râles in one lobe or in both lungs, or extensive consolidation. The persistence of the fever is one of the striking features. Fortunately, a fatal outcome in children is rare. In adults, however, the toxemia quickly gains the upper hand over the merely local symptoms, and puts life in peril in many cases.

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